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VOL. II.—41ST YEAR SYDNEY, SATURDAY, AUGUST 28, 1954 No. 9

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CONGENITAL HEART DISEASE AS SEEN IN COUNTRY PRACTICE.[1]

By CHARLES RICHARDSON,
Ballarat, Victoria.

THE pædiatric problem of congenital heart disease as it affects the country practitioner resolves itself, I think, into definite stages. Firstly, there is its recognition, and then an understanding or knowledge of what is to be done about it, and finally when to act and to have sufficient knowledge of the condition to advise parents on the best course to pursue.

It is therefore necessary for the practitioner to be able, as far as possible, to differentiate the numerous immediate acute post-natal distress states which may be confused with cardiac conditions—the effects of cerebral injuries with hæmorrhage, atelectasis, congenital abnormalities of the gastro-intestinal and respiratory tracts, tachycardias or acute myocardial states, all of which demand recognition and appropriate urgent treatment.

[1] Part of a symposium on "The Pædiatric Approach to Congenital Heart Lesions" held at the annual meeting of the Australian Pædiatric Association, Canberra, April 8 to 11, 1954.

It is, however, in the early months of life and onwards to the early juvenile groups that the problem is most important to the practitioner. Congenital heart malformations are much more common than is generally realized, and deaths from this cause nearly always occur in early childhood.

The teaching that most cases of heart disease in childhood are rheumatic in origin must be revised in the growing knowledge, appreciation and recognition of congenital heart defects and in the probability that a greater proportion of cases are in this latter category. In recognizing a child with congenital heart disease the practitioner must realize that there must be a return to the basic sound principles of clinical medicine, of careful history-taking followed by inspection, palpation and auscultation—history-taking particularly of maternal diet, mode of life or any infections suffered during pregnancy; inspection of the child in a good light and a noting of the colour, the general state of nutrition, the type of breathing, whether it is quiet or noisy or wheezing or whether there is any element of obstruction evidenced by chest retraction. Here it may be necessary to determine if the cause is due to ordinary asthma or if the obstruction is laryngeal or tracheal in type. If on crying or other effort there is present any stridor, one of the rarer congenital vascular anomalies should be suspected and appropriate investiga-

tions carried out. By inspection, too, any asymmetry of the chest is noted or any obvious bulging; palpation may reveal a cardiac thrill. In auscultation the stethoscope returns to its old glory, for in the examination of the child with congenital heart disease there is no substitute. Along with the history and clinical findings it makes possible a reasonably accurate diagnosis in a large proportion of cases. This should be so in the more common defects such as patent ventricular septum, patent *ductus arteriosus* and in later life coarctation of the aorta, but further aids of electrocardiography and fluoroscopic examination should be available to the country practitioner. The presence or absence of right axis deviation would be a significant finding, and fluoroscopic examination would give evidence of heart size and shape and permit a reasonable estimate of the blood flow to the lungs.

Once a congenital heart defect is diagnosed, I would suggest that if cyanosis is present or if there are attacks of pallor or cyanosis or syncope, the country practitioner should refer these children to the cardiac unit for further special investigation, diagnosis and treatment as soon as is reasonably practicable.

Babies and infants who present with the story of failure to thrive or with frequent attacks of upper respiratory tract infection and in whom a cardiac murmur is present provide most cases of patent *ductus arteriosus*. The important feature is that these murmurs are heard most clearly high in the chest and to the left in the second left intercostal space. They may not have the characteristics of the classical machinery murmur at all. It is questionable, I think, whether in infants the murmur of a patent *ductus arteriosus* is purely systolic, and to maintain that a typical continuous murmur should not be expected until the infant is two years old is incorrect. Continuous murmurs do occur in infants much earlier than at two years of age, and, if other clinical features of high pulse pressure or radiological evidence of a large heart and congested lung fields are present, the problem surely becomes one of urgent surgery.

The practitioner must discard all thoughts that these murmurs are due to some compensatory mechanism and that therefore all is well. The extraordinarily varying degrees of cardiac embarrassment due to a patent *ductus arteriosus* makes it impossible to foretell the state of the infant or child's health, even from month to month, and the fact that a patent ductus does not spontaneously close further supports the case for the statement that once a diagnosis of patent *ductus arteriosus* is made in infancy or early childhood it should be closed without undue delay.

The practitioner should have no real difficulties in persuading the parents of these children.

They are probably greatly shocked at times when cardiac surgery is suggested for symptoms not directly referable to the child's heart at all, but after frank discussion and the quotation of so many cases of satisfactory improvement nowadays in these children's height, weight, appetite and general health, there is usually no difficulty.

One note of caution I feel is necessary, and that is that these parents should never be given a completely bright prognosis because of the impossibility of always being sure of an accurate diagnosis and of the fact that cases of recanalization have occurred.

In conclusion, I have been struck by the variations in figures given from different thoracic units of the incidence of early maternal rubella—some give high, others very low, figures. The usual routine at maternity hospitals and in private practice is for mothers to report for advice and treatment for pregnancy at from two to three months.

There is surely a very important relationship between diet in the pre-natal period and reproductive performance, and I feel that intensive investigation should be undertaken into the nutritional status at the beginning of pregnancy and the improvement of the diet of Australian mothers should start before conception has taken place. In this way other factors might be found causing what is now the comparatively common disability of congenital heart disease, and it might be shown that, after all, Nature is not wholly to blame and that the term congenital heart disease may not eventually prove to be a misnomer. To such a dietary investigation country practitioners, if properly organized, could possibly make a worthwhile contribution.

CONGENITAL CARDIAC CONDITIONS IN THE NEWBORN.[1]

By Kate Campbell,
Melbourne.

In considering the subject of congenital conditions in the newborn we must decide, firstly, if a lesion of the heart and/or great vessels is present, and, secondly, if we can, the nature of the lesion.

Is There a Lesion Present?

The History.—The history of the baby's condition at birth will sometimes be a guide. While usually the baby behaves normally, at times it may be slow to breathe and when it does breathe its colour may remain poor. Rarely, the baby may show signs of cardiac failure at birth as in the condition of premature closure of the *foramen ovale*, or in congenital myocarditis secondary to maternal respiratory infection.

The Colour.—In assessing colour it is unwise to be guided by the colour of the extremities. The hands and feet are often like lizard-skin in colour in very young infants owing to vasomotor factors or to cold. The colour of the buccal mucous membrane is a much more accurate indicator. The abnormal colour of the baby is usually the sign which attracts attention. This may be present from birth or may develop later, often in the second week and sometimes much later. I would think most of the babies with gross congenital cardiac conditions whom I see in the early days have a typical lilac or lavender hue at rest. This becomes often a marked, and at times an alarming, degree of cyanosis on crying or with the exertion of feeding or of being handled. Occasionally they become grey and exhausted after exertion. Other infants show marked cyanosis all the time. The distribution of the cyanosis is noted, whether it is more marked in the upper portion of the body or the lower, as this is at times helpful in the diagnosis of the particular lesion. Some babies show no cyanosis but an abnormal pallor. In others the colour is normal.

Dyspnœa.—Though at times dyspnœa is extreme, it is not generally a striking sign and is usually not impressive enough to attract the attention of the nursing staff. Rarely is one asked to see an infant because of dyspnœa alone. Erratic respiration is such a common feature in the newborn that it is frequently disregarded unless it is extreme. In quite grossly cyanotic infants the respiratory rate may be normal. A persistently raised respiratory rate over fifty per minute in the absence of atelectasis or other demonstrable cause is always rather suggestive of a cardiac condition. Dyspnœa is, of course, always more marked after exertion.

The Pulse.—Abnormal pulse rate may be present, tachycardia, irregular pulse or some degree of heart block. The pulse in the newborn varies greatly. In normal infants in the first few days after birth it is often slow, particularly during sleep, and may be 80 per minute. The heart rapidly accelerates to 160 per minute or so during active movement. All pulse rates charted should be sleeping rates. Persistent tachycardia over 140 per minute without obvious cause is always disturbing even in the absence of other cardiac signs.

[1] Part of a symposium on "The Pædiatric Approach to Congenital Heart Lesions" held at the annual meeting of the Australian Pædiatric Association, Canberra, April 8 to 11, 1954.

Feeding Behaviour.—Unusual feeding behaviour is often the first sign to be noticed. The usual story is that the infant begins the feeding satisfactorily but after a short time becomes exhausted. Often the colour becomes worse and there is accompanying dyspnœa and distress, and at times there is marked sweating after feeding. Similar but milder signs may be seen after bathing or handling. By the inexpert these signs are often thought to be due to the universal alibi of infancy—"the wind".

Physical Examination.

In many cases the typical lilac colour suggests the diagnosis at the first glance. Cyanosis, though suggestive, can be produced by many other conditions. Sometimes excessive cardiac pulsation may be visible, indicating an over-acting heart. Bulging of the precordium or asymmetry of the chest is sometimes present, and tachypnœa and laboured respiration may be evident.

On percussion the heart may be found to show clinical evidence of enlargement, and when cardiomegaly is present there is usually some atelectasis, more often on the left side.

On auscultation no bruits may be heard. If a soft systolic bruit is heard it is often impossible to tell whether it is a functional bruit on the way out or an organic bruit on the way in. The organic bruit may be present at birth or may not appear for days or weeks or may not appear till after the neo-natal period. It is very difficult to hear bruits once the child is squirming about or crying, as then the pulse always accelerates. One should endeavour to listen while the child is asleep, folding back the coverings stealthily, sliding up a warm stethoscope chest-piece with warm hands under the baby's clothing. Once the cold air strikes the infant he always begins to squirm about and then to wake. The bruits in these newborn infants usually sound very superficial. A small chest-piece is advisable so that it can be applied satisfactorily to the uneven chest wall. It is difficult to localize the area of maximum intensity without a small chest-piece, as the whole precordium is so small. If the murmur is very loud and the heart is beating rapidly, the bruit may appear to carry over into the second sound. As these infants become older and the rapid rate decreases, the bruit appears to contract to the first sound.

Mild œdema in the newborn in the first few days is so often encountered as to have no special significance. After about the fourth day in full-time infants pitting pretibial œdema is usually pathological and is sometimes found in infants with heart lesions with right-sided heart failure. Hepatomegaly may be present also in such cases. The liver in the normal newborn extends about one inch below the costal margin in the right nipple line. If, however, there is poor chest expansion the liver remains low and the lower edge may then be well below the usual inch from the costal margin, being low but not large. In assessing the size of the liver, therefore, one should take into consideration the degree of chest expansion.

Attacks of anginal pain or of syncope may be present and are often incorrectly regarded as an attack of wind or colic.

Progress.

If the lesion is a severe one the infant fails to gain weight satisfactorily, and many infants of good birth weight lose weight progressively. If the lesion creates little disability the infant may thrive well.

Differential Diagnosis.

The conditions from which cardiac lesions must be distinguished are as follows.

1. Atelectasis must be considered. It is often said that a cyanotic newborn infant with a systolic bruit must necessarily be a subject of congenital cardiac disease. This is not so. A newborn who has any severe degree of atelectasis may develop a loud systolic bruit in the absence of a heart lesion, as proved at post-mortem examination. This bruit disappears when the atelectasis clears. Atelectasis of such severity is usually obvious as indicated by the marked chest retraction and pulmonary signs, confirmed by X-ray examination. A useful crude guide is to note whether the cyanosis becomes less or worsens with crying. Cardiac cyanosis usually becomes worse with crying, while atelectatic cyanosis becomes less. The response to oxygen is also generally helpful. The cyanosis of cardiac defect is unaffected by oxygen, whereas the atelectatic infant shows a definite improvement in both colour and general condition. It is important to observe this response, as otherwise the parents or institution are put to much unnecessary expense for useless oxygen.

2. Bronchopneumonia in the newborn may also produce cyanosis and be accompanied by a systolic bruit which disappears when the pneumonia clears. The colour of these infants improves with oxygen but usually becomes worse when they cry. The history and physical signs in the lungs will usually distinguish these cases, and the X-ray findings will be confirmatory.

3. Diaphragmatic hernia may cause fullness of the left side of the chest with cyanosis and dyspnœa often of a very gross degree. Here again the physical signs aided by X rays will elucidate the condition.

4. Tension pneumothorax of the left side may produce a full left side of the chest with cyanosis and dyspnœa, but the physical signs and X-ray appearances will lead to the diagnosis.

5. Intracranial trauma may be associated with persistent cyanosis due to the accompanying atelectasis or there may be occasional cyanotic attacks of short duration. The raised fontanelle tension, the absence of Moro's sign, associated cerebral symptoms, and, where necessary, diagnostic lumbar puncture usually make the diagnosis clear.

6. The cyanosis of aniline dye poisoning is met with when clothes or napkins have been marked with marking ink and not subsequently boiled. The extreme cyanosis without any distress, and the simultaneous appearance of several cases in a nursery at once, usually direct attention to the correct source.

7. Congenital methæmoglobinæmia is mentioned as a cause of cyanosis, but this is rare. The diagnosis is made by spectroscopic examination.

8. Cyanosis of vasomotor origin should be considered. One sees from time to time infants in whom part of the body is cyanosed and part is normal. When the cyanosis does not extend right across the body in the affected region it is obviously of vasomotor origin. When, however, it affects either the upper or lower portion of the body it may be difficult to distinguish from cases of infantile coarctation of the aorta above the *ductus arteriosus* with a patent ductus, or transposition of the great vessels with a patent ductus.

9. The cardiac conditions with pallor are sometimes mistaken for cases of anæmia or hæmorrhage.

Types of Congenital Lesion.

The types of congenital lesion met with may be any of the following: (i) congenital cardiac defect, (ii) rhabdomyoma, (iii) congenital myocarditis, (iv) aberrant left coronary artery, (v) endosclerosis, (vi) glycogen heart.

Congenital Malformation.

Of these conditions, malformation is, of course, overwhelmingly the most common. Cardiac defect is easily the commonest malformation encountered in the newborn, and is more frequent in premature than in full-time babies. The constructive approach is an attempt to find the cause.

Infection of the mother with rubella at six to eight weeks of pregnancy is a proven cause. It is possible also that other infections may act similarly.

Warkany has shown that if female rats are rendered deficient in vitamin *A* or riboflavin and then become

pregnant, their progeny will show various malformations. I think there is no doubt that parallel conditions occur in humans. If the mother suffers from a vitamin deficiency before she conceives, the fœtus may develop malformations. This possibility will be exaggerated if the mother suffers from nausea and anorexia, vomiting or infection.

Investigation of the nutritional status of the mothers of infants with cardiac malformation will frequently reveal dietetic deficiencies due either to deficient intake or to malabsorption. Riboflavin deficiency can often be traced.

Lawson and DeGaris in Melbourne have directed attention also to deficiency of folic acid in the mother. In one such case of folic acid deficiency under my care, the mother had first an infant who had a fatal persistent *truncus arteriosus*, then an infant who died suddenly in the second week with a rhabdomyoma of the ventricular septum.

These deficient mothers often also have a series of premature births or miscarriages for which there is no obvious obstetric cause and which are almost certainly due to their nutritionally deficient state.

Another possible cause is threatened miscarriage with its reduction of available oxygen to the fœtus if it occurs at the time of cardiac organgenesis. Abortifacients or instrumental interference at this time may also cause cardiac defect.

Any general condition which causes maternal hypoxæmia at this period, for example, severe asthma or bronchopneumonia, may also produce a cardiac lesion.

An hereditary factor appears to present in some families.

Diagnosis of the Type of Lesion.—The types of congenital cardiac lesion met with in the newborn period are different from those encountered in later infancy or childhood. Infants with the very gross and bizarre conditions naturally die earlier and therefore are mainly seen in this age group.

Exact diagnosis of the type of lesion is frequently not possible in the neo-natal period. The different times of closure of the *ductus arteriosus* affect the symptoms. The pressure in the arterial system of the newborn takes some time to become stabilized. This also affects the symptoms and the findings.

In the grossly cyanotic infants who die in the first week or so one suspects such conditions as transposition of the great vessels, bilocular or trilocular heart, persistent *truncus arteriosus*, but various other conditions occur.

Sometimes the distribution of the cyanosis will aid in diagnosis of the particular lesion. If the upper portion of the body is a normal colour with the dividing line at the level of the anterior iliac spines, it indicates a coarctation of the aorta above the isthmus, with a patent *ductus arteriosus*.

If the reverse obtains with the upper portion of the body cyanotic and the lower portion a better colour, it indicates a transposition of the great vessels with a patent *ductus arteriosus*.

Generally speaking, the cyanotic infants are the ones who die in the first two weeks, rarely the acyanotic infants.

It is of interest to note the differing progress of infants who appear equally cyanotic. In some with gross cyanosis the condition improves as the baby becomes older, and in others equally cyanotic there is a steady and often rapid deterioration.

Only the grosser defects cause symptoms in the neo-natal period, those giving less disability being quite asymptomatic.

Physical signs suggesting a cardiac defect are often found unexpectedly on routine examination.

The continuity of observation which permits the following of the natural history of the evolution of signs, symptoms and investigations is valuable for diagnosis and prognosis.

Rhabdomyoma.

Rhabdomyomata may be single or multiple and cause cardiac enlargement. This is a rare condition and causes sudden death without premonitory symptoms. It is frequently associated with other congenital defects of which tuberous sclerosis is said to be the commonest.

Myocarditis.

Myocarditis may occur so early that it must be intrauterine, appearing to be secondary to an upper respiratory infection in the mother. In one case the infant died on the first day and the heart muscle showed typical histological myocarditis with cellular infiltration. The infant was delivered instrumentally and had no post-partum contact with the mother.

Aberrant Left Coronary Artery.

Aberrant origin of the left coronary artery from the pulmonary artery may give evidence of its presence by cardiac hypertrophy and left-sided heart failure during the first month or later. The usual history is of rapid respiration. The infant becomes too tired to suck satisfactorily, so that he takes a long time over feeding and may have leavings. This with vomiting is followed by failure to gain normally. Anginal attacks occur and the heart is found to be enlarged. At the Children's Memorial Hospital, Chicago, F. K. Johnson found this condition in 1·7% of babies with congenital cardiac defects coming to post-mortem examination.

Endocardial Sclerosis.

Endocardial sclerosis is a condition which is attracting an increasing amount of attention. It may be an accompaniment of defects of the left side of the heart, but in the pure condition it occurs alone. It is commoner than the previous condition of aberrant left coronary artery. In Johnson's series it occurred in 10% of the subjects with congenital heart conditions examined at autopsy. This diffuse endocardial sclerosis occurs in the left ventricle and atrium. While the heart is usually much enlarged owing to hypertrophy of the left ventricle, the condition is said by Jesse E. Edwards also to occur without cardiac enlargement, or even with a heart smaller than normal. The thickened endocardium splints the heart muscle, preventing proper emptying in the dilated type and proper filling in the contracted type.

The onset of symptoms may be in the first month, though usually it is later. The symptoms are those of progressive left-sided heart failure, increasing dyspnœa and difficulty in taking feeding, with irritability and failure to thrive satisfactorily. Respiratory infections cause exacerbation of the disease.

If there is marked enlargement of the heart, atelectasis of the lower lobe of the left lung may be present. The condition is fatal.

Glycogen Heart.

Glycogen heart is a rare condition which may produce symptoms of left-sided heart failure soon after birth. There is very gross cardiac enlargement due mainly to enlargement of the left ventricle, and the left-sided heart failure becomes progressive.

Macroglossia is said to occur in one-third of the cases, and hepatomegaly may be present. The only method of making a definite diagnosis is by biopsy of skeletal muscle when glycogen material will be found to be present. The condition is fatal.

Conclusion.

It would appear, therefore, that in the neo-natal period embracing the first four weeks of life, congenital cardiac conditions impose the problems of detection and diagnosis.

The treatment at this age is mainly symptomatic rather than curative.

The question of prevention of the conditions must always occupy the forefront of our minds.

CONGENITAL HEART LESIONS: THE SURGEON'S VIEWPOINT.

By Russell Howard,
Melbourne.

The surgeon's viewpoint can probably best be represented by discussing the various types of case *seriatim*.

Patent Ductus Arteriosus.

The story of the patent *ductus arteriosus* is comparatively old history; one hundred patients have now been operated on at the Royal Children's Hospital, Melbourne. There were three deaths; one was caused by cardiac arrest during induction of anæsthesia and two were due to hæmorrhage, although in both cases the actual bleeding had been controlled before death occurred from cardiac arrest. In one case a recurrence took place and in this ligation has been carried out a second time. Eight patients under the age of eighteen months have been subjected to operation, and of these, four were under four months of age. In these latter cases the patients exhibited very large hearts, a systolic bruit murmur in about the third left intercostal space, a very high pulse pressure and marked failure to thrive. In two of these cases the patent *ductus arteriosus* appears to have been the only lesion, the ductus being of the same diameter as the aorta above it. Both patients have demonstrated almost magical improvement following operation. The other two patients were proved to have a second lesion, probably a septal defect, but their condition appears to have been improved by operation. The first of these four patients developed acute cardiac failure a few hours after operation, and since then we have digitalized pre-operatively any patients with marked cardiac enlargement. These one hundred patients have all been subjected to ligation and none to division of the ductus. In the case in which recurrence took place the ductus was wide and short, and, in retrospect, it is considered that in such instances the ductus would probably be better divided. Practically all patients have shown significant objective and subjective improvement following operation. It is considered that in all uncomplicated cases of patent *ductus arteriosus* in children operation should be performed and that this should be done as soon as the diagnosis can be made with certainty.

Coarctation of the Aorta.

Twelve patients with coarctation of the aorta have been treated by the routine surgical procedure of excision of the coarctation and aortic anastomosis. The anastomosis is thought to be satisfactory in all but one case. In this case operation was performed only a month ago. The aorta between the site of the coarctation and the left subclavian artery, whilts of adequate length, was of narrow diameter. Anastomosis was performed, but the femoral pulses were not palpable until ten days after operation. No aortic graft was available at the time of operation. It would probably have been better to close the thorax as soon as it appeared that the proximal aortic segment was of small calibre, and subsequently to reoperate, to excise the narrowed area and to put in an aortic graft.

In uncomplicated cases operation should not be carried out under the age of seven years. By this time the aorta should be sufficiently developed to provide an adequate post-operative channel for the rest of the patient's life. The sutures used in the anastomosis should be entirely interrupted, as providing the best chance for the anastomosis to grow in calibre with increasing age.

It is considered that in all cases of coarctation of the aorta diagnosed in childhood operation should be performed. If this is done at an early age the risk is small and the results are good. In this age group grafting is required in a very small percentage of cases, as the elasticity of the aorta allows fairly large gaps to be bridged and tearing out of sutures is uncommon.

Aortic Vascular Ring.

Aortic vascular ring is a rare anomaly, presenting in infancy as tracheal obstruction and in adult life as œsophageal obstruction. One case has been encountered and the patient has been successfully operated on. Any infant showing both inspiratory and expiratory stridor should be carefully investigated by radiographic means to confirm or eliminate this diagnosis.

Pure Pulmonary Stenosis.

Coming to conditions requiring intracardiac surgery, let us first consider the case of pure pulmonary stenosis, perhaps associated with auricular septal defect. Brilliant successes are common at all ages from a few weeks upwards in this condition. The universally accepted treatment is division of the fused valvular cusps, most commonly after an approach through the right ventricle.

Auricular Septal Defect.

The operative treatment of auricular septal defect is still in process of evolution. The procedure is hazardous, but it would seem that hypothermia is likely to be a considerable aid to the surgeon in this, as in the surgery of still more difficult anatomical anomalies, such as interventricular septal defects and transposition of the great vessels.

Fallot's Tetralogy.

All anomalies discussed up to this point have one feature in common in their surgical treatment: an endeavour is made to produce a more normal anatomy and the defect is attacked directly. Now we come to Fallot's tetralogy. The surgical treatment of this abnormality has largely consisted in an indirect attack by means of establishing a compensating abnormality — an arterio-venous shunt. It would appear that an abnormal circulation such as is created by this means must give the patient a very limited increase in his life span. In many cases the operation of choice should surely be a direct method of attack on the valvular or infundibular stenosis, as has been devised by Brock.

There are, of course, some cases of Fallot's tetralogy in which the hearts are so grossly abnormal that any attempt at operative correction is impossible and in which death is inevitable in the early months of life. A second group of patients, who survive this period, still present such gross anatomical anomalies that the creation of a compensating arterio-venous shunt is all that is possible in the way of surgical intervention. If life is to be preserved in this group, operation becomes inevitable before the age of three or four years. The patients with less abnormal cardiac anatomy survive the first few years of life. It is these patients who offer the surgeon an opportunity to attack the lesion in a logical way by creating a circulation which more closely approximates to the normal.

Conclusion.

The field of cardiac surgery is one which has only recently been entered and in which progress has been almost incredibly rapid. Who knows what is just round the corner for the present inoperable anomaly?

[1] Part of a symposium on "The Pædiatric Approach to Congenital Heart Lesions", held at the annual meeting of the Australian Pædiatric Association, Canberra, April 8 to 11, 1954.

CONGENITAL OBSTRUCTION OF THE BLADDER NECK.[1]

By Edward S. Stuckey,
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Most text-books of surgery and even of urology pay scant attention to the problem of congenital obstruction of the bladder neck. The bladder responds to obstruction at its outlet by muscular hypertrophy, which may for a

[1] Read at the annual meeting of the Australian Pædiatric Association, Canberra, April 8 to 11, 1954.

time succeed in emptying it completely, but back pressure effects on the kidneys still occur. Sooner or later there is incomplete emptying at each act of micturition, and finally there is chronic retention with overflow incontinence. The bladder wall becomes trabeculated, and multiple diverticula may develop; the ureters become dilated, tortuous and hypertrophied, and the renal pelves and calyces develop gross hydronephrosis. The intramural portion of each ureter may remain normal in calibre for a long time, but finally it also becomes dilated and then free reflux occurs from bladder to kidneys. The effect of back pressure on the nephrons is to produce a slow deterioration of function, with the production of a dilute urine, polyuria, raised blood urea and creatinine content, general failure of growth, and skeletal changes. At any stage infection is liable to occur and it may be impossible to eradicate it even after the obstruction has been relieved.

The clinical picture varies somewhat according to the obstructing agent, its duration and completeness. It is possible, however, to give a general picture of such a child's symptoms. The urinary stream may be slow to start and preceded by obvious straining. The stream may be poor and interrupted from time to time by dribbling, so that the act takes a long time to complete. In later stages there is almost constant dribbling of small amounts of urine from a permanently enlarged bladder. Bladder, ureters and kidneys may all be palpable. When infection supervenes a sudden and serious deterioration occurs in the child's general condition, with perhaps acute retention, toxæmia, uræmia and death.

In investigation examination of the urine and of the blood chemistry should be done first, then excretion pyelography, which in advanced cases will merely establish poor kidney function. Cystoscopy is usually desirable, and cystography, especially micturition cystography, is of great value. Ureteral reflux should be sought for.

General management is important. Uræmia and dehydration must be relieved before the obstruction is corrected. An indwelling catheter may be needed, but may be dangerous as it frequently introduces infection. Sudden decompression may be dangerous to the kidneys. Cystoscopy should be done only when the child is well enough for early correction of the obstruction. In severe cases even suprapubic drainage may fail to prevent stagnation in ureters and kidneys. It may be necessary to perform temporary bilateral nephrostomy.

I wish to emphasize that it is important to observe the act of micturition in all infants and children. These conditions are not being diagnosed early enough, and are present at birth. Therefore I wish to preach the gospel that the newborn infant has not been properly overhauled until some trained person has witnessed the act of micturition. It is not enough to know that plenty of urine is being passed. Normal micturition in the infant is characterized by a certain amount of voluntary effort, soon followed by a sudden steady stream of urine, which starts and stops without preliminary dribbling. Any infant who does not follow this pattern needs urgent investigation.

TYPES OF OBSTRUCTION.

There are three main types of obstruction, none of which is excessively rare. I wish to present a brief case report of each type.

Neurogenic Bladder-Neck Obstruction.

W.P. was three years old when sent into the hospital (May 7, 1951) by my assistant surgeon, Mr. F. Street, who carried out most of his treatment. In infancy he had had a "tumour" removed from his sacral region. He had never achieved bladder control, even by day. When he passed urine he had to strain with his abdominal muscles, and the stream ceased each time he paused for a breath. After micturition copious quantities of urine could easily be expressed suprapubically in a steady stream. His anus was continent, except when the bowels were loose. Sensation in perineum and lower limbs appeared to be normal. Urine was normal; the specific gravity was 1·010. The systolic blood pressure was 120 millimetres of mercury, and the diastolic pressure was 70 millimetres; the blood urea content was 49 milligrammes per 100 millilitres. Excretion pyelograms showed good excretion of dye and no hydronephrosis. X-ray examination revealed a congenital deformity of the sacrum. (See Figure I.) A catheter passed easily and disclosed three ounces of residual urine, and a cystogram showed a large bladder with trabeculation. Reflux was not observed on this occasion. (See Figure II.)

A régime of regular manual expression failed to achieve results, and a bladder neck resection was done on June 20, 1951. The internal meatus would admit a finger, but a fibromuscular median bar was found and removed. After this operation the child could empty his bladder a little better. Several times during the next two years he had attacks of acute urinary infection. Tests on such occasions revealed trabeculation and free reflux of urine to the kidneys, with increasing hydronephrosis, but pyelograms still showed good excretion. Cystoscopy failed to reveal any obstruction. (See Figures III and IV.) A course of "Prostigmin" was tried on one occasion, and later "TEAB" was tried, without improvement of his bladder function. In July, 1953, a further bladder-neck resection was done without improvement. Finally in January, 1954, a bilateral lumbar sympathectomy has been carried out, and the results to date are rather pleasing in that he appears to be able to empty his bladder fully.

Neurogenic bladder-neck obstruction is nearly always associated with demonstrable abnormality of the lumbosacral spine, such as accompanies the various forms of *spina bifida*, but it may follow disease or injury to the spinal cord. *Spina bifida occulta* as an isolated radiological finding, however, is probably not related to bladder defect. Patients may be male or female, and present a picture of which the above case may well serve as an example. There is weakness of the parasympathetic nerve supply, with defective detrusor action and perhaps tonic contraction of the internal sphincter. Often the disability is not apparent until the age of two or three years, the child having apparently had nearly normal micturition earlier in life. It would appear that such patients have passed through a phase in which detrusor action is fairly effective but with spasm of the internal sphincter, and that later the bladder musculature and the sphincter both get tired. A funnel neck in the cystogram is a variable finding. Higgins (1951) states: "In no other type of retention is manual expression of urine so easy, vesico-ureteral reflux so early and so striking, and the bar at the bladder neck so prominent a feature." Wedge resection of the median bar is usually worthwhile, but does not restore normal bladder function. Higgins does not consider sympathectomy of value.

Stenosis of the Bladder-Neck or Marion's Disease.

W.R. had been a patient of mine for inguinal hernia at two years of age, and was brought back to me when he was three years of age with obviously abnormal micturition. He often wet himself by day, having frequency and urgency. He had to strain to make his urine come, and it came in a thin stream often interrupted by dribbling. After micturition a firmly contracted bladder was still palpable, and a soft swelling could be felt in the right iliac fossa. He was admitted for investigation in July, 1952; the urine was normal, with maximal specific gravity of 1·010, and maximal urea concentration of 1%. The blood urea content was 47 milligrammes per 100 millilitres. A cystogram revealed trabeculation of the bladder. (See Figure V.) Excretion pyelograms showed virtually no excretion at two hours. Sounds could be passed without real difficulty, and cystoscopy revealed a small bladder with numerous small diverticula. Retrograde pyelography could not be performed. A suprapubic bladder-neck resection was done on July 23, 1952. The internal meatus was tight and thick-walled. The patient developed a chronic urinary infection and did not seem to be relieved of his obstruction. A month after operation excretion pyelograms showed gross dilatation of the calyces of both kidneys at ninety minutes and elongated tortuous ureters. (See Figure VI.) Cystoscopy was repeated and revealed an unhealed area in the posterior part of the urethra; the ureteric orifices were found, but catheters could not be pushed into either. At this stage it was thought that he had obstruction at the lower end of both ureters, but later a cystogram revealed free reflux into both ureters. He still had recurrent bouts of urinary infection, and it was found that sounds seemed to ride over a ridge at his bladder neck, so on November 26, 1952, his bladder was reopened. A mucosal fold, thought to have developed from excessive granulation tissue, was found and removed. He still seemed

to have to strain to pass urine, and the suprapubic wound took a long time to close, but finally he went home fairly well. When next seen he was passing urine easily, with no accidents by day, and no bed-wetting. Later cystograms revealed no reflux and a good calibre urethra, with a funnel neck. (See Figure VII.) His only trouble now is recurrent right-sided epididymitis, which I feel sure is due to reflux up the *vas deferens*. I have had to tie his *vas deferens* to prevent these attacks. He still has gross hydronephrosis.

This description serves as a good picture of congenital stenosis of the bladder neck. The child may be of either sex, and is usually two or three years of age when the abnormality is recognized, although symptoms must have been present since infancy. It is not till the child is old enough for pants-wetting to be really considered abnormal that the parents become worried.

Transurethral resection is not practicable under the age of four or five years. Young (1950) has devised an "infant punch" for removing a wedge *per urethram*. Higgins recommends a suprapubic approach, with diathermy wedge resection posteriorly, followed by suprapubic drainage. If this is done in time such patients may recover in spectacular fashion, even as regards the hydronephrosis. Results do not seem to be so good in girls.

Congenital Valves in the Prostatic Urethra.

F.G. had been vomiting and failing to thrive since birth, and had been in the Tresillian Mothercraft Home for some weeks when he was admitted to hospital at the age of five months. He had been found to have a large palpable bladder, which he could not empty. Urine was normal; the blood urea content was 80 milligrammes per 100 millilitres. Excretion pyelograms revealed very poor excretion and gross bilateral hydronephrosis. Sounds were passed with no apparent obstruction. Cystograms showed a small bladder with dilatation of the posterior part of the urethra and free reflux into a tremendous right ureter. (See Figures VIII and IX.) It was some weeks before he could be made fit for operation, because of frequent vomiting attacks with dehydration. At operation on October 21, 1953, it was confirmed that he had a dilated posterior portion of the urethra. Incision was carried down into the urethra and two mucosal folds were found and removed. Access was difficult, however, and a catheter still did not pass readily down the urethra from the bladder. He did not do well after the operation and required intravenous therapy several times for attacks of vomiting and dehydration, which were associated with infection. In between attacks he seemed fairly well, but failed to gain weight. He seemed to pass his urine without straining, but its specific gravity was never greater than 1·006 and the blood urea level remained elevated. Three weeks after operation he suddenly collapsed and died. Autopsy was refused.

Congenital valves occur only in the male, and are thought to represent persistence of folds normally present in the embryo. In their usual form they begin on each side at the verumontanum and sweep downwards and laterally, then curve upwards and forwards as prominent ridges in the urethral walls. They may be fused in the mid-line with only a small aperture anteriorly. Such folds have a valve-like action, and are not as rare as used to be thought. Many have been overlooked, even at post-mortem examination. They tend to produce intermittent complete blockage of the stream, but do not offer any resistance to the passage of catheters or sounds from below. Often the renal function is almost destroyed even at birth, and many of these patients die in infancy. The dilatation of the urethra above the obstruction is always demonstrable; but as the bladder neck takes part in the hypertrophy this dilatation may not be observed in a simple cystogram. A cystogram during micturition if obtained will show the characteristic posterior urethral dilatation. Operation is needed in infancy, and a perineal or a suprapubic approach may be used. The dilated urethra must be opened and the folds found and removed. If this is done properly obstruction should not recur, but the renal function may be so poor that the infant may not recover.

In conclusion, the end results of surgery in bladder-neck obstruction are disappointing mainly because the condition is not diagnosed early enough.

Legends to Illustrations.

FIGURE I.—Case I. Plain skiagram showing marked asymmetry of sacrum associated with *spina bifida*.

FIGURE II.—Case I. Micturition cysto-urethrogram showing dilated bladder with small bladder neck pouch.

FIGURE III.—Case I. Excretory pyelogram at thirty minutes showing dilatation of lower end of ureters.

FIGURE IV.—Case I. Excretory pyelogram at thirty minutes showing early hydronephrosis of kidney.

FIGURE V.—Case II. Cystogram showing gross trabeculation of bladder, with diverticula.

FIGURE VI.—Case II. Excretion pyelogram at 120 minutes shows delayed excretion, gross hydronephrosis and megaureters.

FIGURE VII.—Case II. Micturition cysto-urethrogram. Wide bladder neck and no urethral obstruction now.

FIGURE VIII.—Case III. Cystogram showing reflux into enormous right ureter, displacing trabeculated bladder to the left.

FIGURE IX.—Case III. Micturition cysto-urethrogram showing marked dilatation of bladder neck and prostatic urethra.

DEAFNESS DUE TO RUBELLA.[1]

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IN this paper the epidemiology of rubella in Australia is discussed with special mention of the factor of isolation. As statistics of rubella infections are not available, the epidemiology of measles in Australia is compared with that in other countries.

Measles and rubella are infections characterized by short incubation periods, short periods of infectivity, and a solid and persistent immunity after infection. Such diseases, as is known from field observations and suggested by theory, have great difficulty in maintaining themselves in any population. For if disease is efficient in passing from a sufferer to a susceptible person then the whole population of susceptibles may be exhausted in a few generations. On the other hand, if the disease is less efficient, there is always a definite probability of its dying out before the susceptibles have been exhausted. The usual text-book model of infectious diseases should be modified, so that the population is not represented by one homogeneous unit but by a number of "isolates" or sub-units, which are relatively isolated from one another. Thus the family forms one type of isolate. An infant in arms in a family will usually contract infectious disease only from another member of the family. In English cities this leads to a distinctive epidemiological pattern with epidemic waves every second year. Most adults and older children being immune, in an epidemic year the toddlers and younger children are infected; in the next year there will be susceptible infants but an insufficiency of young children to carry the disease from one isolate to another. In the following year, however, there will be enough susceptibles among the toddlers and younger children to spread the disease. This epidemic behaviour is different from that holding in Australia, until recently at any rate. Lancaster (1952) gave the deaths per year in the different States from 1890 onwards in a table, which showed that the epidemics were more widely spaced, especially in the smaller States, than the English. Smaller aggregates within the State, if analysed separately by notifications (Cumpston, 1927), show even longer intervals between epidemics. Measles, in fact, seems to have died out in some areas completely and then to have been reintroduced after many years.

[1] Part of a symposium on "Problems of the Deaf Child" held at the annual meeting of the Australian Paediatric Association, Canberra, April 8 to 11, 1954.

The bearing of this on the epidemiology of rubella is clear. Australia may be expected to have had epidemics of rubella and then long periods of quiescence; under these conditions a large proportion of females will have had their infection in adult life. More particularly, females born in the decade 1911 to 1920 would have an excellent chance of reaching adult life without experiencing the disease, as there was little, if any, rubella between 1924 and 1937 in Australia. Perhaps rubella was reintroduced from abroad in 1937, as was held by Scholes (1944), who noted that 1914, 1923 and 1937 and later years were years of high rubella incidence. Lancaster (1951) was led to study the date of birth of the institutional deaf by a study of census reports. He found that 1898 and 1899, 1916, 1924 and 1925 and the more recent years 1938, 1940, 1941, 1943 were times of birth of unusually high numbers of children later to be admitted to institutions.

As a rapid method of comparing deafness experience in different countries, inquiries were directed to other countries. New Zealand (Lancaster and Pickering, 1952) has had two great epidemic periods—one in 1899 and another beginning in 1938—for births of the deaf. Unpublished data suggest that Iceland has also had an epidemic of births of the deaf in 1941. The inquiry failed to show any epidemic in institutional data from Italy and from Sweden for certain years. The data were not sufficiently unequivocal for the years after 1936; but Ivstam (1951) has shown that rubella has been causing deafness since 1936 in Sweden. England has had an experience similar to that of Sweden. In the earlier years there was no evidence of epidemics of the deaf, but since the beginning of the war there have been epidemics. The experience of England and Sweden can be probably best interpreted as due to a change whereby an increasing proportion of individuals have been passing through childhood without contracting infectious diseases. Possibly non-specific factors such as hygiene, family size and so on are of importance (Lancaster, 1952). Similar figures suggest that the American population has had an experience intermediate between the Australian and that of the great land masses.

It may be that in the future in Australia, with greater population and with more rapid means of transport to and from other countries, rubella will become endemic and that there will be sporadic cases of rubella deafness without great epidemic episodes as in the past.

The analysis above makes it unnecessary to postulate any special feature of the rubella virus in Australia. It has been the population that has been distinctive; the epidemiology of rubella deafness in Australia and New Zealand, two isolated countries, has been similar.

Acknowledgement.

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THE EARLY RECOGNITION OF DEAFNESS IN CHILDHOOD.[1]

By V. L. Collins,
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Early recognition of deafness is today being emphasized for a number of reasons. Firstly, the whole educational approach to the deaf child has changed—there is no longer a place for the term of deaf-mute or deaf and dumb, and sign language has little place in teaching today; the emphasis is upon the use of hearing aids where possible and upon early training in normal speech, and in speech (lip) reading and the development of language so that the deaf child can take a reasonably normal place in society and communicate with those around him. To obtain the best results in speech training, early recognition of deafness and early training are essential. Oral speech teachers would like to start training the mother of the deaf child as early in the child's infancy as possible, and certainly would like to commence the training of the child by eighteen months of age. Secondly, early recognition of deafness is essential if mismanagement and behaviour disorders are to be avoided. Thirdly, early recognition of deafness from naso-pharyngeal conditions and middle ear disease will permit of earlier treatment and often the prevention of greater hearing loss (Lewis, 1950; Davey, 1952).

Degrees of hearing loss vary. Mild degrees of hearing loss are common; total deafness is rare, and there is usually some degree of hearing in the severely deaf child. In this paper, little reference will be made to the commoner mild degrees of deafness in which there is a 15 to 30 decibel loss, not requiring the use of a hearing aid. Other speakers will make further reference to this group, for early recognition of deafness here with early treatment of the ear, nose and throat conditions commonly present undoubtedly plays some part in the prevention of more severe deafness.

Here, it is planned to emphasize the early recognition of deafness in those children with severe deafness or partial deafness—in children who have a hearing loss greater than 25% or greater than 30 to 35 decibels. Mistakes have been made frequently in this group owing to failure upon the part of medical practitioners to appreciate the symptoms and behaviour of the deaf child. Children with a severe hearing loss from birth have reached the ages of ten and twelve years before the handicap was recognized.

A review of 58 children examined at the Royal Children's Hospital, Melbourne, during the past twelve months has been made. All these children were severely or partially deaf. All the 58 either needed a hearing aid or required admission to a special school because of deafness. Altogether 63 patients were examined, but three patients have been excluded from the series because of inadequate detail, and two had severe unilateral deafness only.

In this series there were 27 severely deaf and 31 partially deaf children. Table I shows the age at which deafness was first recognized, and the number in each group.

It will be seen that it was possible to recognize the severely deaf before they reached the sixth year of life, and that 19 out of 27 were recognized between the ages of two and four. Recognition of the partially deaf was considerably delayed. The majority—20 out of 31—were recognized between the ages of three and seven years, and a number of them much later. Though a number of these children had acquired deafness during childhood, the majority had been deaf from early in life or from birth.

Any attempt to define ætiology in such a group of patients is certain to be incomplete. In reviewing the literature, one soon becomes aware of the deficiency of knowledge upon the causes of deafness. In the present

[1] Part of a symposium on "Problems of the Deaf Child" held at the annual meeting of the Australian Pædiatric Association, Canberra, April 10, 1954.

series it is evident that bone conduction tests when possible upon younger children, and follow-up tests upon certain other children, would be essential at a later age to clarify the cause.

It will be seen that in almost 50% of the cases (Table II) there is no obvious cause and a congenital origin seems the most likely. Some studies (Bakwin and Bakwin, 1953) include a smaller figure (30%) of cases present in early life in which there is no obvious congenital cause and which appear to be pre-natal or peri-natal in origin. In

TABLE I.
Age when Deafness Recognized.

Age in Years.	Number Severely Deaf.	Number Partially Deaf.
0 to 1 ..	2	0
1 to 2 ..	4	0
2 to 3 ..	12	0
3 to 4 ..	7	6
4 to 5 ..	1	0
5 to 6 ..	1	7
6 to 7 ..	0	7
7 to 8 ..	0	0
8 to 9 ..	0	4
9 to 10 ..	0	2
10 to 11 ..	0	1
11 to 12 ..	0	1
12 to 13 ..	0	3
Total ..	27	31

these reviews Rh incompatibility has been a cause of 4% of deafness, but does not occur in the groups above. Infections are much the same as here with meningitis 3% to 4%, maternal rubella 4%, *otitis media* 15%. The three

TABLE II.

Ætiological Factor.	Number Severely Deaf.	Number Partially Deaf.	Total.
Maternal rubella	2	1	3
Familial	2	0	2
Meningitis	3	0	3
Ear, nose and throat cases (*otitis media*, adenoids and measles)	2	13	15
Birth trauma	3	2	5
Dihydrostreptomycin	1	0	1
Mumps	1	—	1
Total with apparent ætiological factor	14	16	30
? Congenital	13	15	28
Total	27	31	58

cases of purulent meningitis above were in infants under twelve months of age, and one each of meningococcal, pneumococcal, and *Hæmophilus influenzæ* type.

The presenting symptoms fall into two main groups. The parents sought advice either because they thought their child was deaf or because of a speech disorder without suspecting deafness (Table III). A few presented because of backwardness or behaviour problems. Though not the main complaint, behaviour disorders were a prominent problem in 18 of the 58 children. It will be seen that in the presence of severe deafness the parents suspected deafness fairly early. With partial deafness, a complaint of speech defect at a later age was the most common complaint. In one child aphonia was the main symptom causing anxiety.

How might deafness be recognized at an earlier age in either of these groups and particularly in the group of the partially deaf? A better knowledge, firstly of certain aspects of hearing, and secondly of the developmental behaviour of the deaf child, would undoubtedly help. Also, in the light of this knowledge, a review of some of the mistakes which have been made should be of assistance.

In the average person the hearing range is between 20 and 16,000 cycles per second—a cycle being a complete vibration. Human speech usually falls between 250 and 4000 cycles per second, and a ready understanding of speech calls for a reasonable balance of hearing within this range. A hearing loss has been found most frequently at 256 cycles per second. A 30 decibel loss at 2000 cycles per second will cause a greater handicap than a similar loss at a higher or lower tone (Davis, 1947).

TABLE III.
Presenting Symptoms.

Onset Symptoms.	Severe Deafness.	Partial Deafness.	Total.
Suspected deafness	19	12	31
Speech disorder	6	14	20
Behaviour problems	(8)	1 (10)	1 (18)
Backward	2	4	6

Hearing may be normal in the lower range but severely defective in the higher range, causing what is called high tone deafness (as occurs in nerve-type deafness). Hearing, therefore, may be normally sensitive for lower pitched sounds, and so a hearing defect is not considered. High tone deafness causes a loss of hearing of consonants which fall between 1000 and 4000 cycles. "S" and "f" for instance are lost, and so speech can be severely affected.

A serious loss in the mid high range makes it difficult to distinguish differences in consonants, and such words as bed, led, said and dead sound very much alike. It is apparent that such an impairment represents a serious interference with the acquisition of normal speech habits. Fairly good hearing for test frequencies up to 1000 cycles per second, but considerable or severe impairment for frequencies above this, is common in congenital or nerve-type hearing impairment.

A loss of hearing in the lower range interferes with an appreciation of vowel sounds which are heard in the range up to 1500 cycles per second.

Pure tone audiometry has made possible an accurate assessment of the hearing loss. An estimation of the degree of loss is measured in decibels. The decibel is the unit of intensity or loudness. Theoretically the decibel is the smallest change in intensity which the human ear can detect. The intensity of range for the human ear—that is, the range between the point where sound just becomes audible and the point where sound is so intense as to be painful—is about 130 decibels. A hearing loss of 30 to 35 decibels (approximately a 25% loss) or more constitutes a partial hearing defect and is the lower limit at which a hearing aid is used.

Very quiet conversation ranges between 45 and 50 decibels, loud conversation between 60 and 65 decibels. A loud shout at one foot is 110 decibels. An aeroplane engine close by is 120 decibels. A passing aeroplane is 100 decibels (Davis, 1947). A child with a 75% to 70% hearing loss, therefore, may hear an aeroplane passing overhead or a train whistle. This observation often leads to the mistaken belief that the small child's hearing is normal, and nothing is done about checking his hearing as a result.

There are three types of deafness: (i) Perceptive or nerve-type deafness. (ii) Conductive deafness due to

diseases of the middle ear, lymphoid obstruction and accompanying respiratory infections, and occasionally to allergic conditions. This is the most favourable group for active treatment. (iii) Mixed types.

In order to appreciate the problems of recognition of deafness in an infant, it is necessary to understand the developmental behaviour of a severely deaf child. From Table I it will be seen that few of these children have been recognized as deaf under two years of age, and many were nearly three years of age or over. Why should this be so? Perhaps the main reason for overlooking deafness is lack of realization that most deaf babies pass through the same behaviour stages as normal babies—they play with rattles and enjoy them, and they laugh and babble. Vocalizing occurs at the usual age. Deaf babies gurgle and coo in normal fashion. Between the ages of nine months and eighteen months they frequently appear to be developing normal speech with such words as "Mum Mum", "Dad Dad", "bub bub", "ah ah", "la la", "oh a wa wa". It is only when this vocalizing stage continues without improvement or vocalizing ceases completely between the ages of eighteen months and two years, and further speech fails to develop, that earlier suspicions of deafness, owing to failure to respond to calling and other household noises, may again be roused. Even then, the fact that the child has responded to a passing aeroplane or a train whistle may convince the parent (and doctor) that the child is just slow with speech.

Three children, severely deaf from meningitis before the age of six months, illustrate this. All were saying "Mum Mum" and "Dad Dad" by about twelve months of age.

T.D. suffered from meningococcal meningitis at the age of six months. When seen in the follow-up clinic at twelve and fifteen months of age he was found to be developing normally, using words, "Dada" and "Mum Mum", and could undoubtedly hear certain sounds. He was discharged. The mother brought him back at the age of three and a half years because of his failure to speak. He had temper tantrums when his wishes were not understood and he did not respond to speech. He was referred to the audiometrist and found to have a 70% hearing loss.

A little girl, N.J., was initially brought to hospital at the age of one year and nine months because the parents thought she was hard of hearing. There was a history of maternal rubella at the sixth week of pregnancy, and evidence of a congenital heart lesion. The doctor stated that she was not deaf because she appeared to hear and understand certain words—for example, "don't touch", and she said "Bub", "Mum" and "no". Ten months later the doctor commented that deafness was no longer a worry, but she was still saying only single words. The parents again attended when she was four and a half years of age, saying only a few words. They stated that she did not appear to hear them, but heard the telephone. She watched faces, screamed, and became very excited when visitors came, and concentrated poorly. She was found to have an 80% hearing loss in both ears (Figure I). She made an excellent response to a hearing aid, took it to bed with her, and tried to imitate all speech sounds. She was a bright, intelligent girl.

Gesell and Amatruda have listed certain signs suggestive of deafness in infants and young children in the developmental order in which they are most likely to appear.

1. *Hearing and comprehension of speech.*
   - General indifference to sound.
   - Lack of response to spoken word.
   - Response to noises as opposed to voice.
2. *Vocalisations and sound production.*
   - Monotonal quality.
   - Indistinct.
   - Lessened laughter.
   - Meager experimental sound play and squealing.
   - Vocal play for vibratory sensation.
   - Head banging, foot stamping for vibratory sensation.
   - Yelling, screeching to express pleasure, annoyance or need.
3. *Visual attention and reciprocal comprehension.*
   - Augmented visual vigilance and attentiveness.
   - Alertness to gestures and movement.
   - Marked imitativeness in play.
   - Vehemence of gestures.
4. *Social rapport and adaptations.*
   - Subnormal rapport in vocal nursery games.
   - Intensified preoccupation with things rather than persons.
   - Inquiring, sometimes surprised or thwarted facial expression.
   - Suspicious alertness, alternating with co-operation.
   - Markedly reactive to praise and affection.
5. *Emotional behaviour.*
   - Tantrums to call attention to self or need.
   - Tensions, tantrums, resistance due to lack of comprehension.
   - Frequent obstinacies, teasing tendencies.
   - Irritability at not making self understood.
   - Explosions due to self-vexation.
   - Impulsive and avalanche initiatives.

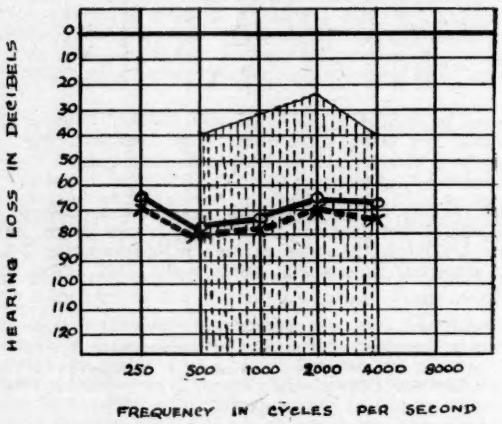

FIGURE I.

Audiogram of patient with rubella deafness. Shaded area represents range within which hearing loss is sufficiently severe to require a hearing aid. O = right, X = left.

In cases of severe deafness, parents have often suspected deafness for some time, but have done nothing about it until a more serious disability became apparent. Doctors still say: "He may be deaf, but there is nothing to be done about it until school age and then he can go to the deaf and dumb school." In very severe deafness, parents often notice lack of hearing early. "He does not hear me come into the room." "He makes no response to calling." Where the child hears average noisy sounds it is easy to overlook deafness.

H.M. was found to be severely deaf at the age of six months. She was brought to hospital because she made no response when a nurse dropped an object making a loud noise when she was four and a half months old.

D.G. was found to have severe deafness at the age of two years three months. The parents stated that she failed to take notice of speech at the age of nine months. They saw a doctor, who said nothing was wrong. At twelve months of age they saw an ear, nose and throat specialist, but no special advice was given. Finally they saw another specialist, who recommended attendance at hospital. When the child was seen he was very aggressive, banged his head frequently, and lacked concentration.

L.G. was found to have a severe hearing loss at the age of three years four months. He said "Mum" and "Dad" at the age of twelve months and then stopped saying these words. The parents persisted in trying to make him speak, but he would put his hands over their mouths and push them away.

M.P. was found to have severe deafness at the age of two years three months. His mother reported that he did not respond to calls and watched lip movements. He had frequent attacks of screaming and head banging.

F.P. was found to be severely deaf (hearing loss left ear 80%, right ear 90%) at the age of three years and four months. He was brought to hospital because of poor speech and his aggressive behaviour towards other children. It had been noticed that he did not respond to commands, and made his wants known by signs. He had been a quiet baby and could hear aeroplanes. Within two weeks of the fitting of a hearing aid and the commencement of oral training and guidance to the mother, he had acquired four words, commenced to speech read, and accompany gestures by voice. Play with other children showed marked improvement.

P.S. was first recognized as being severely deaf, with no response to pure tone audiometer, at the age of five years and two months. He was first taken to a specialist at the age of eighteen months because he was developing slowly, he was generally hypotonic and was regarded as suffering from cerebral palsy. He began to walk at the age of two and a half years. He saw the specialist again at three years of age because speech was not developing, but his poor speech was regarded as a sign of backwardness, the same as

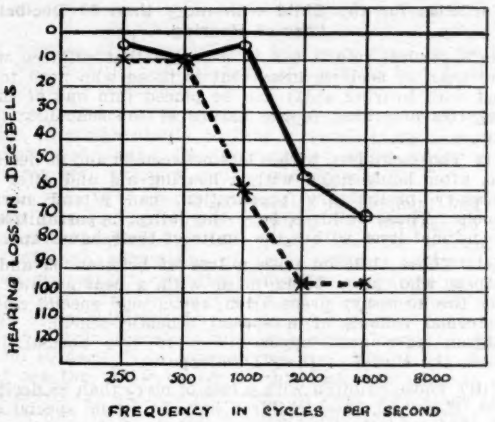

FIGURE II.

Audiogram of girl (M.O'C.) showing high tone deafness. O = right, X = left.

the slow passing of his other "milestones". The mother finally sought further advice because he did not respond to verbal instructions but obeyed gestures. He was found to be deaf and of normal intelligence, and still to suffer from a weak and hypotonic muscular system.

The moderately deaf or partially deaf child may present a different picture, and often, as mentioned above, his speech will be the main problem or he may appear slow to learn, be vague and appear to lack concentration, or appear frankly backward. These patients form the group in which most mistakes have been made in the past. The loss or misuse of consonants in cases of high tone deafness or a monotonal voice immediately lead the experienced observer to consider deafness. There are other children with poor speech, however, in whom the diagnosis of deafness is much more difficult. Mental retardation or hearing defect or both together may cause the speech defect. On the other hand a child with dysialia may not be deaf or mentally retarded and present with the same speech defect as with high tone deafness. The correct decision can be made only if audiometric testing is done in all cases of speech defect.

O.M. was found to have high tone deafness at the age of nine years and four months when she was referred to hospital because of poor speech and enuresis. She had passed the usual "milestones" normally, had talked at two years of age and used sentences at the age of two and a half years. Her mother reported that her voice was high pitched and she gabbled. She substituted m's for t's, and j's for n's. She was unable to pronounce "s" or "th". She said "bakinette" for bassinette. She was backward at school and had difficulty in reading and spelling. She had a 95 decibel loss at 2000 and 4000 cycles in the left ear, and a 60 to 70 decibel loss at the same tones in the right ear. She was unsuitable for the usual hearing aid on account of normal hearing at 1000 cycles per second (see Figure II). With special training her speech and auditory discrimination gradually improved and enuresis stopped.

W.P. was found to have high tone deafness at the age of ten and a half years. She was referred because of backwardness. Her mother said she could hear and understand, she would talk in simple phrases to other children but only in single words to parents and adults. She was taken to a doctor in another State at the age of five years, and he said she was deaf and dumb, but a specialist denied this and recommended tonsillectomy and removal of adenoids. After the operation she seemed to improve. She went to school at the age of five years and was still in the baby class when referred five and a half years later. The mother thought she was wilful and disobedient. The child was very withdrawn and wept continually when first examined. She was found to understand instructions only if demonstrations were given. Her speech was indistinct and high pitched and lacked inflection. She had a 45 decibel loss at 500 cycles and a 90 to 100 decibel loss at 4000 cycles (see Figure III).

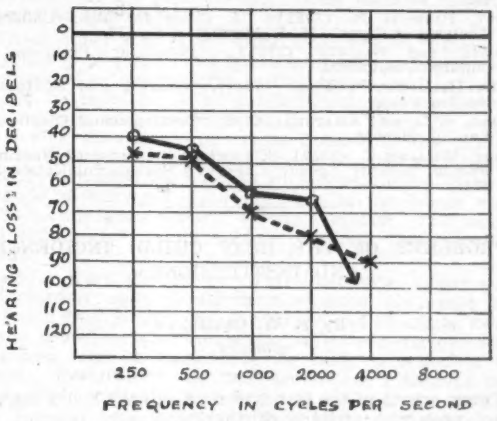

FIGURE III.

Audiogram of girl (W.P.) showing high tone deafness. O = right, X = left.

R.D. was found to have a 60% hearing loss in both ears at the age of twelve years. He was brought to hospital because he was regarded as being very backward at school. He had suffered from discharging ears on and off since infancy. His mother first thought the patient was deaf at the age of seven years, but this was denied at another hospital and also by several doctors. Prior to the onset of deafness he had passed the usual "milestones" early—walked at the age of eleven months, had early speech with good sentences at seventeen months of age, and his habits were clean by the age of fourteen months. He was of normal intelligence.

B.W. was found to have a 30% hearing loss at the age of eight and a half years. He was referred to hospital because he had been playing truant and stealing. He was regarded as backward at school, with poor speech. His home conditions were poor. After he had been to the ear, nose and throat department, and tonsils and adenoids had been removed, there was complete restoration of hearing.

A fitting conclusion to this brief review is a comment by Hardy (1952): "There is no longer any need for the child to arrive at the age of 5–6 years with a hearing impairment that is undiagnosed, unmeasured, untreated and uncompensated for by early guidance and training." It is no rare thing nowadays for a child, aged four years, with severe hearing loss to be well on the way towards a good vocabulary and a happy adjustment to the family and the world around him.

Summary.

Emphasis has been placed upon the earliest possible recognition of deafness in childhood, so that training in

oral speech and speech reading may be commenced at the most appropriate time in the second year of life and the maximum use made of hearing aids, so that the mother may be guided in the management of her child as early in infancy as possible, and so avoid mismanagement and behaviour disturbances; and so that ear, nose and throat conditions may be treated as early as possible. Certain aspects of deafness in early childhood are reviewed and illustrated by a review of 58 children with severe and partial deafness seen at the Royal Children's Hospital, Melbourne, over the past twelve months.

Acknowledgements.

I should like to express my indebtedness and thanks to Mrs. E. R. Lawton, oral speech teacher and audiometrist at the Royal Children's Hospital, Melbourne, for considerable help in reviewing the patients' records. Mrs. Lawton has done a great deal to educate the medical staff of the hospital in the needs and possibilities of the deaf child.

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PROBLEMS OF THE DEAF CHILD: INCIDENCE AND INSTITUTIONS.[1]

By F. W. CLEMENTS,
Sydney.

THREE events in the last fifteen years have had a marked effect upon the attitude of the community towards the problems of the deaf child and upon the nature of the services which the medical profession and the community are able to provide.

The rubella epidemic of 1937-1943, which produced a large number of deaf children, somewhere between six and ten times the numbers in the pre-epidemic years, focused the attention of the medical profession and the public in Australia on the needs of the deaf child to an extent that had not happened before. The development of pure tone audiometers has provided instruments which make it possible to screen large numbers of children in a more satisfactory way than does any other method hitherto in use. This advance has made possible the detection of minor degrees of deafness at a much earlier age than was formerly possible.

The perfection of a small hearing aid which can be adapted for quite young children has considerably altered the nature of treatment. I think we would all agree that ideally, the children suffering from any illness or defect should be treated in circumstances that will enable them to live lives which depart as little as possible from those of the rest of the child community. Until the development of satisfactory hearing aids this was not possible for deaf children. Although the hard-of-hearing child sat in the front of the class, he often missed much, not only of formal education, but also of the give-and-take by which most children learn to fit into the community. The more deaf child was doomed to a somewhat monastic existence in a special deaf school where he too often had the minimal opportunities to learn to live a normal life.

I want to see how far the Australian community has availed itself of these newer developments in the handling of deaf children. In the preparation of some of this material I have drawn upon the reports of the Commonwealth Acoustic Laboratories.

Facilities for the Child with more than 30 Decibels Loss of Hearing.

As a general rule it can be accepted that children with more than 30 decibels loss (that is those who need to be fitted with hearing aids) can be placed into one of three categories according to the nature of the schooling they need.

(i) Those children with a loss between 30 and 65 decibels who, after being fitted with a hearing aid and after the necessary preliminary preparation, can attend normal schools. Those children have the fullest opportunities to live normal lives within the limits of their handicaps.

(ii) Those children with a loss of between 65 and 85 decibels who, after being fitted with a hearing aid and after the necessary preparation, can attend special classes in normal schools or a special acoustic school. These children have good opportunities to live normal lives within the limits of their handicaps.

(iii) Those children with a loss of more than 85 decibels. Most, if not all, these children have to attend special deaf schools, where the teaching is mainly oral; here the opportunities to live normal lives are limited.

A survey by the Commonwealth Acoustic Laboratories found the distribution of 174 deaf children born 1944 to 1948 living in New South Wales as set out in Table I.

Table I shows that too few children attend special acoustic classes and too many attend deaf schools. Many of the children contributing to this surplus are children who could attend special acoustic classes if these were available. Such classes are provided in the metropolitan areas, but hostels are needed in which country children could live while they attended these classes. Tasmania is the only State which had provided such a service at the time of writing. The only alternative for these children is for them to be admitted as boarders at the deaf schools.

[1] Part of a symposium on "Problems of the Deaf Child" held at the annual meeting of the Australian Pædiatric Association, Canberra, April 8 to 11, 1954.

TABLE I.

Distribution of Children Born 1944 to 1948, According to the Type of School being Attended.

Degree of Hearing Loss.	Normal Primary and Secondary Schools.	Pre-school—Central Acoustic Laboratory.	Acoustic Classes—Education Department.	Spastic Centres.	Deaf Schools.	No School.	Total.
30/65 decibels	48	3	6	6	1	3	67
65/85 decibels	5	0	13	4	20	1	43
More than 85 decibels	1	2	3	3	52	3	64
Total	54	5	22	13	73	7	174
Numbers which might be expected to attend schools according to audiometric tests	48	5	39	—	52	—	144

Children with 15 to 30 Decibels Loss of Hearing.

In addition to the children with 30 or more decibels loss of hearing there are those who have a hearing loss of from 15 to 30 decibels. As a rule none of these, at this stage of deafness, require a hearing aid. These children can be detected in mass surveys made with pure tone audiometers.

A survey of children living in Canberra, by officers of the Commonwealth Acoustic Laboratories, returned the results set out in Table II.

TABLE II.

	Number of Children Tested.	Percentage of Children Failing Screenery Test in One or Both Ears at 15 Decibels Limit.
Primary schools	3043	7·35
Secondary schools	652	4·57
Pre-school children ..	136	4·4

A number of these children had more than 30 decibels loss and required hearing aids; the natural history of the remainder is unknown.

A routine school medical examination in New South Wales, in which ability to hear a watch is the screening method used, found the incidence of hearing loss to be 1·4% for both primary and secondary schools.

A number of longitudinal studies in England and the United States of America on children with between 15 and 30 decibels hearing loss have shown that the hearing in a significant percentage of these children deteriorates unless appropriate remedial measures are started as early as possible. In Australia we need to know much more about these children; for example, we should know how many children there are with a hearing loss of between 15 and 30 decibels in each community. We need to know what are the major causes of this loss, and what is the natural history of these children. In what percentage does hearing deteriorate if not treated adequately? What are the criteria for treatment?

CROSS INFECTION IN THE NEO-NATAL NURSERY.[1]

By KATE CAMPBELL,
Melbourne.

Classification.

CROSS INFECTION in the newborn nursery presents two main problems: (i) that of the introduction and (ii) that of spread of infection. Broadly speaking, there are two groups of infection: (*a*) the episodic or sporadic group, in which the main emphasis is on the prevention of the introduction of infection, and (*b*) the continuing or endemic infection, in which the main emphasis is on the prevention of spread of infection.

The Episodic Infection.

In general the episodic infections are more easily dealt with than the continuing group. They consist mainly of (*a*) the gastro-intestinal infections; (*b*) the respiratory infections, and (*c*) the infectious diseases—for example, chicken-pox, measles *et cetera*.

Sources.

The sources may be: (*a*) the mother, (*b*) labour ward and nursery personnel, (*c*) extrahuman agents.

[1] Part of a symposium on "Cross Infection in the Hospital" held at the annual meeting of the Australian Pædiatric Association, Canberra, April 8 to 11, 1954.

The Mother.—The mother is often the source of gastro intestinal infection. About the end of pregnancy mothers are so accustomed to discomforts and bowel irregularities that gastro-intestinal disturbances are often ignored. During labour the disturbances are often thought to be due to enemata or to the castor oil which is frequently given. It should be a practice to question every mother admitted about diarrhœa or vomiting over the previous week or two, and nurses should be instructed to report any abnormalities of the stool. If a positive history or abnormal stool is present, the mother and the baby are isolated separately and the baby is given prophylactic treatment. In this way we have been able to prevent nursery infections on several occasions. If the infant had been transferred to a nursery before the mother's infection was reported, the nursery was closed to further admissions till all the infants had been discharged. The particular mother's infant was transferred to isolation.

Upper respiratory tract infections in mothers on admission are often disregarded. They are a frequent source of infection of their own babies, who then spread the infection through the nursery. These mothers should be treated as infectious.

These upper respiratory tract infections in infants can be followed by serious and fatal conditions, such as bronchopneumonia or myocarditis. Any babies with a coryza should be isolated. It is not sufficient to keep them in the nursery and barrier-nurse them. There is usually little risk that frank infectious disease in the mothers will be ignored and they therefore rarely start an infection. Any mother admitted with an infectious disease should if possible be delivered elsewhere than in the labour ward, so as to minimize contacts among the staff. The mother and infant are usually transferred from the midwifery wards.

Labour Ward and Nursery Personnel.—The labour ward and nursery staff should be instructed to report any deviation from health, however minor, and should be aware of the danger of their conveying infection. Labour ward personnel may be responsible for early infection of the infant. Respiratory and gastro-intestinal infections may be carried, particularly if a mouth catheter for sucking out is used. Such catheters should have an adequate trap in the glass bulb to protect the baby. Nursery staff are very frequently the origin of respiratory infections. It is surprising to find how casually these infections are regarded, so that one may meet a nurse with an upper respiratory tract infection in charge of one's most precious premature infant. Some physicians also are very light-hearted about the potential infective power of their respiratory infections.

Gastro-intestinal infection may also originate in nursery personnel, though less commonly, as most people have a healthy respect for "gastro" infections. The most usual error for a nurse is to mistake the onset of a gastro-intestinal infection for an upset due to some article of diet.

Technicians and domestics, though theoretically a source of infection, have so little direct contact with the babies that danger from them is probably minimal.

Extrahuman Agents.—The possible avenues of infections from other than human agents are the laundry, ingredients of artificial feeding, feeding bottles and utensils, breast milk brought in from outside the hospital, and such rare occurrences as infected fæcal matter from cats, mice and cockroaches. Adequate control of laundry procedure and supervision of laundry workers are necessary to block infection from this source.

If there is any infective condition, particularly gastro-enteritis, in the house from which expressed milk is brought, it should be discarded till the infection is over. The milk itself can be boiled and sterilized, but the possibility that the staff who come in contact with the person from the infected household may themselves become infected constitutes a danger.

Once an infection is established the usual procedures are instituted. This necessitates three nurseries with three separate staffs both day and night.

(i) The isolation nursery to which the affected infant is removed. This should be a self-sufficient unit, with its own sterilizer, stethoscope, stationery *et cetera*. Bowls of antiseptic fluid should be of adequate strength and removed as necessary. There should be a sufficient supply of small, easily disposed of hand towels. Arrangement should be made so that feedings are delivered to a table outside the nursery, and the isolation staff makes no contact with staff from clean nurseries.

(ii) The original nursery in which the infection occurred. All the babies in this nursery are at risk and no fresh babies are admitted till the epidemic is past.

(iii) A clean nursery into which all new cases are admitted till the epidemic is over.

The Continuing Infection.

The continuing infection is the difficult type to eradicate. It is favoured by overcrowding, understaffing and a poor general level of nursery hygiene. The commonest type is sepsis, while thrush appears to be endemic in some nurseries. Rarely is gastro-intestinal infection a continuing problem in neo-natal nurseries.

Sepsis of a severe type can continue on and off for years in a nursery, being almost always due to *Staphylococcus aureus pyogenes*. Once established, its ubiquitous spread renders its eradication difficult. There is no doubt that the rather exuberant exhibition of antibiotics has encouraged the emergence of resistant strains. All the nursery *Staphylococcus aureus* strains which I encounter are penicillin-resistant.

The protean manifestations of neo-natal sepsis are well known, ranging from conjunctivitis or small pustules to fatal septicæmia.

The Labour Ward.

In attempting to eradicate nursery sepsis one must start first in the labour ward. Where eye drops are used as a routine measure it may occur that infection of the drops may cause a purulent conjunctivitis or rhinitis. These drops should be kept in proper eye-drop bottles and prepared freshly each week. It is wise to have them cultured from time to time.

The boracic lotion used for eye swabs should be investigated. Very crude and queer methods of preparation are uncovered at times.

The freshly cut cord is very frequently exposed to infection and may be the point of entry in cases of septicæmia. The infant at birth is often wrapped in a merely clean napkin. This he soon kicks off, so that the raw end of the cord comes into direct contact with blankets or bunny wraps which are surgically unclean. Moreover, it is a common sight to see infants being subjected to their first bath shortly after birth with the cord floating submerged in the bath water.

The infant should be received into a sterile wrap until such time as the cut end of the cord is protected. Routine bathing is best dispensed with.

Mucus catheters and those used for the intranasal administration of oxygen are in general treated poorly. Mucus catheters are usually sterile when first used, but thereafter are left lying about anywhere. Instead of sterile material to receive the ejected mucus being used, any piece of material is used. The baby's end of the catheter is often allowed to become contaminated by the end which has been in the operator's mouth, and so carries a real risk to the infant.

Intranasal oxygen catheters are frequently not sterile on introduction and are often kept dangling at the side of an oxygen cylinder.

In the Nursery.

It is impossible to eradicate neo-natal sepsis while overcrowding continues. There is no doubt that dispersal of infants is the biggest safeguard, and this is best effected by nursing babies at the mother's bedside—the so-called "rooming-in" régime.

I investigated conditions in a communal nursery and found that each baby was attended by six different nurses as well as the mother in a day, and was handled twenty-eight times. Compare this with the bedside baby who is attended by his mother and one nurse, and handled seven times a day. In those hospitals where both systems are practised the infection rate is definitely lower among the bedside babies than among those nursed in communal nurseries.

When communal nurseries are used it is wise to have them as small as possible and partitioned. There should be three feet between the centres of adjacent cots. It is not unusual to see the babies' cots packed side to side round the periphery of a nursery, available space in the middle of the room being left empty. The recommended area of five feet by six feet for each cot is rarely practicable at present. Nurseries should be reasonably equipped. There should be hand-basins with foot or elbow operated taps.

Each infant should have his own individual locker. All infants should be cot changed, and communal baths and change tables should be dispensed with. Each infant should have his own bowls for oil and swabs, and it is desirable that he should have his own thermometer.

A clean piece of tissue paper should be placed on the scales to weigh each baby.

Blankets should be covered by sheets and should not come in contact with the baby's skin. Any piece of clothing or bedding which falls on the floor should be discarded and sent to the laundry.

Soiled napkins should be hygienically disposed of into covered sterilizable containers. Nurses should wash their hands after changing infants, and it is advisable that the nurse who does the changing should not do the feeding.

The food room must be adequately supervised in regard to the making up of feedings and the sterilizing and storing of feeding bottles and utensils.

Nurses.

The best insurance against neo-natal sepsis is a good nursery sister. It is most necessary that all nursing staff be adequately instructed in nursery hygiene, and in the need for the early detection, reporting and isolation of nursery sepsis. They should also have a good understanding of the manner in which any sepsis in the staff can introduce infection. This applies especially to mother-craft nurses, who are being increasingly employed in community hospitals.

It is well known that the nose is the big reservoir for *Staphylococcus pyogenes*, and in the past I felt that masks properly used were a safeguard. I find now that it is impossible to ensure that the staff uses masks properly. Members of the nursery staff everywhere handle masks in a way they never handle the naked nose. I have therefore decided to dispense with masks.

The resistance of *Staphylococcus pyogenes* to different antibiotics affords a rough method of typing them, and I think it is wise to have nurses swabbed routinely for *Staphylococcus pyogenes* before they take up nursery duty in the same way as they are swabbed for hæmolytic streptococci before taking up labour ward duty. Any attendant with manifest sepsis or significant *Staphylococcus pyogenes* in nose or throat culture should be excluded till free from infection.

The nurses should be sufficient in number (one to twelve for healthy babies, one to six for sick babies) and should be well fed.

Babies.

A close watch should be kept on the babies. As regards care of the cord, I have been afraid to follow the naked cord technique in the presence of nursery sepsis. We have used sterile forceps for dressing the cord, and 1 in 1000 monacrin in spirit if the cord looked at all doubtful.

All clothing in direct contact with the babies' skin should be boilable.

Paronychia is often lightly dismissed as having been caused by the infant sucking his fingers. It is forgotten that merely sucking the fingers cannot produce infection unless the *Staphylococcus pyogenes* is present in the infant's mouth. Routine swabbing of the nose and throat of other infants showing no lesions will often yield the staphylococcus at these times.

Other common minor manifestations are conjunctivitis, rhinitis, and mild omphalitis. Spreading upward from the omphalitis we may notice indications of infective phlebitis of the umbilical vein. It is these minor manifestations, so often treated as trivialities, which keep the infection going.

More marked infection may appear as impetigo, pustules and boils. As the infection becomes more severe, cellulitis of an alarming rapidity and intensity may be encountered. Periostitis, Smith's arthritis, staphylococcus pneumonia and empyema, pericarditis, periostitis, meningitis, pyæmia occur, and at times a septicæmia of frightening intensity and suddenness. I think there is no doubt that some cases of kernicterus in premature infants are due to septicæmia.

It is frequently not appreciated that septicæmia in the newborn is often afebrile and this may be a reason for delay in the institution of treatment.

Any infant with sepsis, no matter how minor, should be immediately transferred to an isolation ward with a complete staff who care for no other babies. This is generally a very unpopular move with the minor grades of sepsis, but unless it is rigdly enforced the infection in the nursery continues.

No matter how minor the sepsis, it should be actively treated. Many people are loath to treat these minor infections actively because they fear to make the strain insensitive to antibiotics. This fear is misplaced, as, firstly, the longer the infection lasts, the greater are the possibilities of spread, and secondly, each minor infection may develop into a major one in a particular infant. There is also a real danger that the infant will infect the mother's breast. Indeed, recently it was stated to me at an infant welfare sisters' conference that the commonest cause of failure in breast feeding today was breast abscess following sepsis in the baby.

The appropriate antibiotic is given. In the case of cellulitis *et cetera* we have found the recommended reduced dosage, namely, six milligrammes of aureomycin per pound of body weight per day, adequate. In two cases of fulminating septicæmia we have given 25 milligrammes per pound intravenously, divided into two doses. (One baby recovered, the other died.)

The administration of antibiotics is often accompanied by the occurrence of buccal thrush. Vitamin *B* complex and acetomenaphthone are given to all babies receiving antibiotics.

It is necessary to ensure that all bedding, including blankets and mattresses, is adequately sterilized after any case of sepsis.

When sepsis is present in a nursery, routine circumcision should be avoided on any infant, whether showing evidence of sepsis or not.

An infant who is in isolation for sepsis should not go to the breast: firstly, because isolation is not adequate if the mother is free to come in and out of the isolation ward, and secondly, because of the risk of breast infection in the mother.

Thrush infections in nurseries may go on year after year. The fault is often the practice of covering mouthpieces of bottles with a napkin instead of something sterile to act as a cap. Failure to appreciate the high infectivity of the condition is also another factor, so that isolation of the babies is not practised.

The two big practical difficulties in controlling nursery infection in most hospitals today are (*a*) the lack of any satisfactory isolation ward or wards, and (*b*) the absence of a pool of extra nurses to staff these wards when the emergency of infection arises.

It is extremely difficult to make lay people realize that isolation beds should be kept empty for emergency. They look upon all idle beds as so much money lost, and while one sympathizes with hospital administrators in their financial difficulties, it is necessary that isolation beds equal to at least 10% of the nursery beds should be permanently available.

It would, I think, be helpful in ensuring this if this association were to draw up standards of accommodation and care for the newborn and distribute them to doctors and hospitals.

Conclusion.

In conclusion it would appear that the main means of prevention of infection are: (*a*) an informed and numerically adequate nursery staff free from infection, who practise a high level of nursery hygiene; (*b*) reasonable equipment for the nursing of newborn infants; (*c*) the avoidance of overcrowding; (*d*) adequate isolation facilities.

THE PHYSICIAN'S WORRIES IN RELATION TO THE OLDER CHILDREN.[1]

By ERIC SIMS,
Adelaide.

THE risk of cross infection amongst older children in hospital is probably as great as it is amongst babies, but it tends to be viewed more complacently because the outcome is not usually so disastrous. Nevertheless, the many hazards that attend a second, hospital-brewed, infection superimposed upon the primary illness constitute a major problem for pædiatricians. Some of these infections are difficult to control whilst children's hospitals are constructed mainly on the large ward plan. The fact that a child has been in contact with a specific infectious disease may not be mentioned by the parents, or even known to them, when a child with an acute respiratory illness (for example) is being admitted to hospital. Even the admitting officer may not think of the possibility of measles, or notice the Koplik's spots. Only after a day or so in the open ward does the appearance of a rash make the diagnosis of measles apparent, and by then the damage is done. Atypical cases of various communicable diseases also may catch anyone napping, and patients may be admitted to open wards; and the susceptible young nurse in the incubation period of a disease may remain on duty until a minor epidemic is under way amongst her charges. Indeed, members of the kitchen or nursing staffs may spread an epidemic of diarrhœa amongst children when they themselves have only a minor bowel upset from an enteric infection.

Cross infection in hospitals also involves many less dramatic illnesses which may produce considerable morbidity. These include the various upper respiratory infections that lead to sinusitis, otitis, bronchitis *et cetera* with chronic sequelæ. Skin sepsis also can lead to quite major complications. The prevention of all these shame-making events is, up to a point, merely a question of applying bacteriological knowledge that has been available for years. Only inertia, staff shortages, and bad architecture permit much of it to occur at all.

MODES OF INFECTION.

I have always liked Mitman's classification of the various modes of infection into:

(i) Aerial infection, in which the air is the vehicle of spread, either immediately by droplets, or more tardily by droplet nuclei and infected dust.

(ii) Direct infection—or "contact"—in which there is direct touching between infector and infectee.

[1] Part of a symposium on "Cross Infection in the Hospital" held at the annual meeting of the Australian Pædiatric Association, Canberra, April 8 to 11, 1954.

(iii) Indirect infection, in which there is contamination of intermediate objects such as fomites, insects, or food, and the conveyance of the pathogens by these vectors, to the infectee, perhaps quite remotely in time and place.

And to these should be added, for completeness:

(iv) Spread by insect vectors which are acting as intermediate hosts. This need not be considered further here.

Mitman advocated this classification to obviate the confusion that often surrounded the use of the term "direct contact", and to include the modern concept of the potential infectivity of the atmosphere itself indoors.

Aerial Infection.

Under the heading of aerial infection must be included all respiratory diseases, including those specific exanthemata that are spread via the air.

Selection of Cases.—Their control in a children's hospital will depend, firstly, on adequate care in selection of cases for institutional care by the patients' own doctors. No child of any age should be sent into a hospital unless it is manifestly impossible to nurse him at home (for medical or social reasons), or unless the nature of the illness makes in-patient investigation imperative. Often, one feels that a practitioner has referred a child to hospital because he cannot be bothered to pursue further the problem privately, or because he wants to pass on the responsibility of decision. Indeed, I think I have done it myself.

Admitting Procedure.—Once, however, the fact has been accepted that the particular child does really need hospital care, the next stage is careful consideration by an experienced admitting officer. I am not sure that I enthuse over the Medical Research Council's recommendation that the child immediately be attacked with swabs in every orifice, but at least a carefully taken history and a thorough inspection of the patient are essential.

Isolation.—Then, if the slightest possibility of a communicable disease exists, the child should be referred at once to isolation in a single cell, or to a small ward reserved for the particular illness.

Here we immediately come up against the problem of hospital construction and organization—the large open wards, the practice of grouping children by ages or clinics rather than by diseases, the insufficiency of plumbing, *et cetera*. Nevertheless, the Medical Research Council's recommendation that nearly half the beds in a children's hospital should be in individual cells (with walls extending from floor to ceiling) must stand as an ideal before us. The patients in such cells must be nursed by full isolation, "barrier" ritual wherever indicated—the glossiest glasshouses are futile if the attendants are not impeccable in their techniques.

Ventilation.—The problems of ventilation remain of first importance in the control of aerial infection and sometimes are insuperable when one tries to subdivide old wards by high partitions. "Air-conditioning" may be a useful substitute for sufficient windows, but often consists only of air-cooling rather than of air-sterilizing, and it may not really effect adequate air exchanges. Adequate space, too, is essential, but this requirement presupposes that the hospital is big enough to cope with the demands the community makes upon it, without overcrowding. This is seldom the case. Until one can build an entirely new hospital, one must often compromise between the ideal and the practicable. Indeed, in many old wards I would rather see numerous hand basins than numerous partitions, and a thoroughly trained, germ-conscious staff than ultraviolet lamps. Perhaps a hand basin between all beds would at least ensure a moderate gap between patients!

Face Masks.—Even face masks can become a facade behind which many septic sins are committed in a false sense of security. They become doubly farcical when they do not include the nose, or when they are worn all day, and are doffed and donned, sometimes inside out (as witness the lipstick), when occasions appear to demand. A thoroughly contaminated mask can become a reservoir for the constant reinfection of the hands each time the slipping fabric is readjusted. Under these circumstances, one can sympathize with those who maintain that the mask is a mixed blessing, and that the accent should be on other methods of prevention.

Dust Suppression.—Dust suppression by oiling floors is widely advocated, and is obviously helpful on appropriate surfaces. I cannot help feeling, however, that this is a method that has its best application on wooden floors, with crevices, *et cetera*. It is not so safe—and perhaps not so necessary—on linoleum or rubber floors; but in any case dry sweeping and dusting are obviously to be avoided. The oiling of blankets is also advocated to minimize the spread of infected particles during bedmaking. Washable blanket-envelopes also may serve the same purpose, and mattresses must obviously be protected against contamination as well.

Nursing Staff Problems.—A sufficiency of nurses must be provided to carry out the work (as much as possible) in an unhurried fashion, with full attention to barrier techniques, hand washing *et cetera*. Division of labour so that the same girls are not required to do both "dirty" and "clean" duties is axiomatic, but here one comes up against problems of arranging nurses' rosters, of the human desire (that even nurses have) to help one another, of peak demands on thinly spread resources, *et cetera*. Even so, the ideals are known, and must be striven for. We, on our part, can set an example of careful technique; disseminate a basic bacteriological knowledge amongst nurses; immunize nurses and encourage them to report promptly when they are sick; and relieve them of any unvital duties—clerical and domestic—which take up time that could be better spent in nursing. Much of the charting and recording that nurses do is theoretically for our benefit, but often smacks strongly of the traditional chore, the necessity for which has not been reviewed by doctors for a generation.

Night Nurses et Cetera.—"Floating" nurses, with running duties in several wards, must be discouraged. There is, however, another nurse whom one often overlooks—the night nurse. On two occasions in the past, we have had minor outbreaks of diphtheria in wards at the Adelaide Children's Hospital, and on both occasions the cause was found in the throats of the night nurses, who were thought of only in a last desperate effort to trace the focus of infection. So, when confronted with an epidemiological problem, think of the septic sins committed under the cover of darkness by an overworked, lone hand who is even now not always given the support she needs.

Primary Pulmonary Tuberculosis.—A burning question in Adelaide recently has been the infectivity or otherwise of primary pulmonary tuberculosis. It has been the time-honoured practice at the Adelaide Children's Hospital to nurse children suffering from this infection in open wards amongst other children, and, as far as we had been aware, no harm to others had resulted. However, a year or two ago a baby who was finally shown to have tuberculosis (but who, incidentally, did not react to the Mantoux test himself) was probably responsible for the Mantoux-conversion of 18 nurses who were in contact with him. We still continue to admit patients with pulmonary tuberculosis to open wards, but we realize that this is not ideal from any point of view. The hazards of secondary infections in the tuberculous child himself, as well as the (possibly remote) risk to others, have made us discuss the desirability of a children's sanatorium. This has not yet been attained, and opinions still differ on the form it should take.

Visitors.—The question of visitors is perhaps a controversial one. It is manifestly unwise for a parent who has a cold, or is in any way potentially infectious, to visit his or her child in hospital. With this proviso, and with reasonable parental cooperation, I do not believe that daily visiting increases the risks of cross infection. The important transmitters from bed to bed are the nursing and medical attendants themselves. The contact the parent has with other children in the hospital should be minimal, and the emotional benefits of the visit to the child are far too important to be ignored. This, of course, particularly

applies to the younger children, but even the older ones are often better able to make the necessary adjustments after returning home from a stay in hospital if they are constantly kept in touch with the family in this way. Here, again, a compromise between the conflicting interests of the bacteriologist and the psychologist must be achieved. Nowhere in the Medical Research Council's memorandum on "The Prevention of Cross-Infection in Hospital" did I find reference to the child's reactions to the techniques recommended. Perhaps at times a happy child with a coryzal sniffle is a better sight than an aseptic child with a lonely sniffle. Indeed, we are told that grief can have as devastating an effect on well-being as can infection.

Control of Outbreaks.—Finally, when we are confronted with a ward outbreak of infection, various techniques must be put into effect to limit or put down the outbreak. This is, perhaps, more the province of the clinical pathologist to discuss, so I will confine myself to two aspects. Firstly, wherever practicable, the ward should be closed for all new admissions, for a sufficient period of time. Even so, I have been impressed with the long periods over which chickenpox can smoulder on in a ward; and, in any case, it is usually not possible to resist for long the demands for relief of the other wards which are beginning to overflow and beget their own problems.

Mass Immunizations and Treatment.—The second aspect is the prompt mass use of such immunization procedures as are available, and indicated by the particular infection. Gamma globulin, to prevent the wide spread of measles in a ward, and perhaps chemoprophylaxis if a streptococcal infection is occurring, are two examples. However, two of our biggest bugbears at present are the antibiotic-resistant staphylococcus and *Pseudomonas pyocyanea*. Much morbidity is resulting from these organisms, and we have few chemical resources to control them.

Direct Infection.

The control of direct spread of infection depends on the intelligent cooperation of the attendants, and should not be a big problem. Again handwashing and the prompt treatment of any infection, general or local, in any member of the staff are the main requirements.

Indirect Infection.

Indirect infection, of course, includes mainly the diarrhœal illnesses, due to Shigella and Salmonella organisms, or to the various other pathogens, bacterial or viral, that have been incriminated. The tetralogy of F—food, fingers, flies and fæces—is the usual story. Here again the clinical pathologist may be able to help us, but the points I have already made apply equally well to these diseases. Careful assessment in the admitting department and the referring of the child to an isolation unit reserved for such cases—or to bedside isolation if necessary—are of first importance. This will often mean that children are sorted together on clinical rather than bacteriological grounds, with the consequent risk of mixed infections, but this can be kept at a minimum by careful technique, separation of nursing duties *et cetera*.

It is our practice at the Adelaide Children's Hospital to admit all children with diarrhœa to a small open ward reserved for that purpose, and mixed infections contracted within the ward are practically unknown. The thorough and prompt disinfection of all contaminated fomites, and the disposal of excreta with the minimum of splash, followed by sterilization of all containers and the washing of hands are all essential. The control of flies and cockroaches by appropriate insecticides also is important in the control of indirect cross infection in hospital.

Summary.

1. Considerable morbidity results from hospital infections amongst children even after the infant age group.

2. No child should be sent for admission to a children's hospital without careful assessment by his own doctor of the risks to the child himself, and to others.

3. The nature of the illness should be ascertained as far as possible by a reasonably senior admitting officer, and should in some instances be the deciding factor in the child's destination within the hospital.

4. Children's hospitals should have adequate space, adequate isolation accommodation, adequate ventilation, adequate plumbing, and adequate staff.

5. Nursing staff should have some basic bacteriological instruction; they should be relieved of unnecessary duties, and encouraged to report promptly when sick. Absolute separation of "dirty" and "clean" duties is essential.

6. The medical staff should set a good example.

7. Face masks, improperly used, may be a snare.

8. Visitors are at the most a minor factor in cross infection, and with certain provisos should be encouraged.

9. The whole problem of cross infection in hospitals could be greatly reduced by the determined application of long-established principles. Only inertia prevents this.

CROSS INFECTION IN THE HOSPITAL: THE PÆDIATRIC SURGEON'S PROBLEMS.[1]

By K. B. Fraser,
Brisbane.

Introduction.

In discussing "cross infection in the hospital" one must have clearly in mind what is meant by the term. From the surgical angle I am taking this to mean inflammatory conditions of any kind conveyed either directly or indirectly to surgical in-patients from all and sundry with whom they come into contact. In the bacteriological sense it means then the acquisition by a patient of pathogenic microorganisms not present on his admission to hospital.

The many and varied types of infection conveyed from hand to mouth in the out-patient department cannot, in the time available, enter into consideration here. Neither will it be possible to discuss the dangers inherent in the use of syringes in hospitals.

While many detailed laboratory investigations on the subject of cross infection have been carried out, I must confess at the outset that, apart from what I have read of the work of others, my opinions are based largely on clinical impressions, not supported by scientific data. As an offset to this, however, I have had an opportunity of working for many years in two children's hospitals where, by force of circumstances, conditions in many ways are diametrically opposed.

There is no doubt that, while the problem of cross infection is an important one in any hospital, it is doubly important in a children's hospital, where we are dealing with a particularly vulnerable patient population.

At the present time in Australia many hospitals are at a disadvantage because of old-fashioned buildings, poor equipment and shortage of staff, but these drawbacks can be largely overcome by an awareness of the factors involved in cross infection. This is one aspect which, in my opinion, has not been given enough prominence in the past, either in student teaching or in the hospitals themselves.

Looking backwards, I cannot but feel a sense of guilt that I have not made greater efforts to encourage a campaign of awareness in the hospitals in which I have worked. This necessity for "awareness" applies not only to the medical staff but also to the nurses, lay staff, visitors, and even to the patients themselves.

From the pædiatric surgeon's point of view the question of cross infection is important in two directions. There is,

[1] Part of a symposium on "Cross Infection in the Hospital" held at the annual meeting of the Australian Pædiatric Association, Canberra, *April 8 to 11, 1954.*

firstly, the cross infection of patients in the ward before and after operation, whereby they may contract either one of the exanthemata or any of the other easily transmissible bacterial or virus diseases: this can well be called "non-surgical" cross infection of surgical cases. Secondly, there is the cross infection as a result of which burns, operation wounds of skin or mucous membrane and skin graft sites may become contaminated — so-called "surgical" cross infection.

Of these the first—that is, non-surgical cross infection—is more common and in my experience is far more difficult to cope with.

Non-Surgical Cross Infection in Surgical Cases.

Non-surgical cross infection, which is often serious enough in the older age groups, is particularly devastating in children under two years of age.

Upper respiratory tract infection is a troublesome post-operative complication at any age, for it delays recovery and prolongs hospitalization. Fortunately in very few cases nowadays does the infection seem to go on to bronchopneumonia; possibly this is because most surgeons unhesitatingly defer operation if the anæsthetist reports any sign of respiratory infection.

Streptococcal tonsillitis can, on occasion, spread rapidly through a crowded surgical ward. The common infectious exanthematous diseases are, of course, always with us, as is infectious hepatitis, and these diseases can, in the prodromal period, cause cross infection among surgical patients.

In spite of all these possible sources of non-surgical cross infection, however, gastroenteritis still remains the greatest hazard. This applies particularly to bowel infection in the napkin-age group, which is, without doubt, the greatest menace of all as far as the pædiatric surgeon is concerned. Nothing is more galling than to find that a patient recently operated on for pyloric stenosis, intestinal atresia or hare-lip repair has been whisked away to an infectious disease ward because of loose offensive bowel actions.

The old so-called epidemic summer diarrhœa of Queensland is now fortunately a thing of the past. In 1908, with an infant mortality rate of 70‰, diarrhœa accounted for 42% of these deaths; in 1946, with a mortality rate reduced to 29‰, diarrhœa accounted for less than 10% of the deaths.

Infantile diarrhœa, however, still remains a smouldering fire in the community, and tends to flare up in a ward in the most aggravating way; in this connexion it is interesting to note that 3·5% of children in Brisbane are Salmonella carriers.

In the children's hospitals in my State in recent years the offending organism has been found to be generally either a Salmonella or a Shigella, with *Escherichia coli* (*a*-coli) less frequently encountered. M. J. and I. M. Mackerras, Rubbo, Rogers and many others have written extensively about the vagaries of the different species of these offending bacteria, and further detailed discussion of the causal organisms is beyond my province.

Mackerras's investigation into recent outbreaks of gastroenteritis in the Brisbane Children's Hospital showed clearly that important links in the chain of cross infection were: (*a*) the presence of subclinical gastroenteritis infection in patients admitted for some other reason altogether; (*b*) infection carried from baby to baby by the nursing staff, almost certainly due to contamination occurring during the changing and handling of napkins; (*c*) contamination of nail brushes in the ward; (*d*) presence of carriers among the nursing staff.

In addition, Rubbo in Melbourne in 1948 showed conclusively that dust could be a potent factor in gastroenteric cross infection. How, then, can one hope to cope with this menace to carefree surgical nursing of infants and young children?

Ideally, of course, all patients with suspected bowel infection should be immediately removed from a children's hospital where surgery is done, but this is usually impracticable and is often impossible because of the specialized nursing required in the unfortunate event of post-operative infection in major surgical cases. An important aim, however, is in all surgical cases to nurse children under one year of age in cells or in cubicle wards restricted to surgical cases only.

As soon as one talks of nursing children in cubicles, an apparently irreconcilable clash of interests comes to mind. To achieve economy of effort from the nursing angle children are best nursed in long, open wards; to minimize cross infection they are best nursed in individual compartments. This problem has fortunately been overcome to a great extent by the modern type of cubicle ward, with glass partitions, built in bays, in which the children are separated from each other but are under general observation, and in which the construction is such that good nursing care with a normal-sized staff is possible.

FIGURE I.
A modern cubicle observation ward, showing one of the five six-bed bays.

Very few, if any, hospitals in Australia are lucky enough to have all their wards built in this way, but adequate beds of this type should be provided to cope with all cases in which suspicion arises, either on the patient's admission or during the course of treatment. However, while we all agree on the importance of good ward construction, bricks, mortar and glass alone do not make a good hospital, and there is no doubt that a high standard of ward hygiene by everyone concerned is the sheet anchor in the control of all types of cross infection. To emphasize this, Rogers has shown how quickly a cubicle becomes contaminated, how long it remains contaminated, and how communal articles in a ward can be blamed for intercubicle spread of infection.

Well-maintained barrier nursing will overcome most of the disadvantages of poor accommodation and even a great many of the hazards of overcrowding. It is important also to remember that careful screening of intended patients will minimize the number of patients admitted who go to constitute a pool of cross infection, either surgical or non-surgical. Non-urgent suspects can be deferred, and any suspected patients who must be admitted can be "filtered" through an observation ward.

Careful organization in the use of time and labour-saving devices will allow the limited nursing staff at present available in most hospitals more time to devote to their essential nursing duties. An example of the time and energy that can be wasted was provided by one particular

hospital, not long ago, when pedometer measurements showed that the nursing staff walked seven and a quarter miles in distributing one meal!

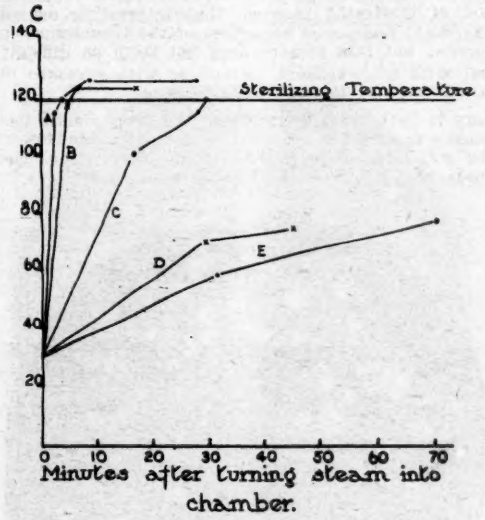

FIGURE II.

Thermocouple records of temperature graphed against time during recent tests on five different autoclaves. Autoclaves A and B are satisfactory; autoclave C is effective only if the time lag is allowed for; autoclaves D and E are defective.

It seems that the necessary high standard of ward hygiene cannot be achieved unless all concerned have impressed on their consciousness the hazards which care-

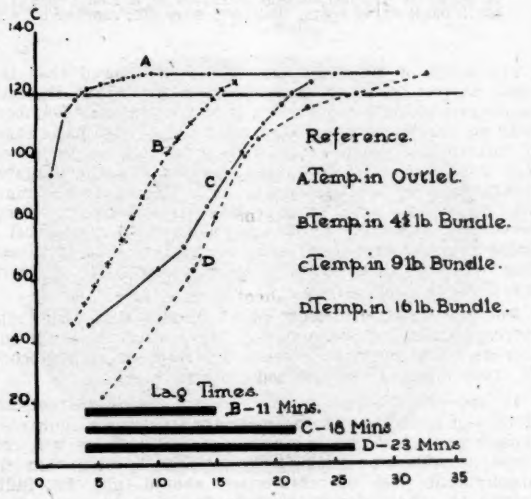

FIGURE III.

The danger of making autoclave bundles too large and too heavy.

lessness brings in its train; there is no better way to do this than to give periodic demonstrations in practical bacteriology. These can be made to demonstrate in a dramatic way the ease with which pathogenic organisms can exert their malign influence in a ward. It is also a most important thing for each member of the staff to know of the precautions required of all other members, whatever their particular duties may be. This helps to drive home the lesson that momentary individual carelessness can nullify, in an instant, the continued painstaking efforts of many others.

FIGURE IVA.

A sneeze not quite completed showing the expulsion of atomized secretion as revealed by the high-speed camera. (By courtesy of the American Association for the Advancement of Science and Marshall W. Jennison, "Aerobiology", 1942.)

It is not my intention to dwell on the many details concerned in control of ward cross infection; these have been very clearly laid down in the Medical Research Council Memorandum Number 11 (Revised Edition, 1951) dealing with the control of cross infection in hospitals. There are, however, one or two points I should like to stress.

FIGURE IVB.

A sneeze behind a mask showing the great reduction in the number of droplets passing through the mask. (By courtesy of the American Association for the Advancement of Science and Marshall W. Jennison, "Aerobiology", 1942.)

I have always been impressed by the minimal amount of dysenteric cross infection which occurred in military hospitals during the Second World War, although they were operating often under very difficult conditions. I have come to the conclusion that this was due to four main reasons: firstly, the fact that the nursing staff was a fully trained one; secondly, that the patient population was an entirely

adult one, with the elimination of the napkin menace; thirdly, that the Army machinery allowed rigid ward hygiene to be easily enforced; and, fourthly, that all pans and fæces were immediately boiled in very carefully fly-proofed surroundings, rather than treated with disinfectant, which is an ineffective and outmoded method.

The direct and indirect inferences to be drawn from these circumstances could well be borne in mind when cross infection control measures are being established in any children's hospital.

Probably in all wards, but most certainly in infants' surgical and gastroenteritis wards, duties should be so arranged that no nurses changing napkins have anything to do with handling food, and *vice versa*.

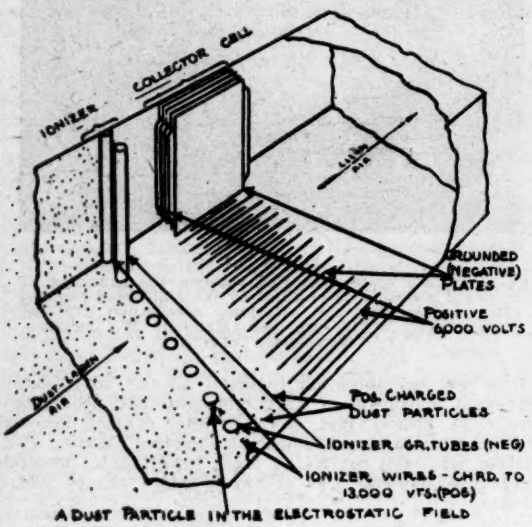

FIGURE V.

Diagrammatic representation of an electrostatic filter showing how positively charged dust particles are deposited on the negatively charged collecting plates.

Singer and Ludford have stressed the importance of impressing on the nursing staff the necessity to report any digestive upset, however slight this upset may be.

Surgical Cross Infection in the Wards.

To the casual observer the incidence of cross infection of wounds may nowadays seem negligible, but in my experience a careful survey of all types of wounds in a surgical ward still discloses an appreciable degree of cross infection, often of no serious import but on occasion so severe that quick and satisfactory recovery is prevented.

Many of the points already mentioned in the prevention of non-surgical cross infection apply equally forcibly when surgical cross infection is considered. Here again it is impossible to describe in detail the many precautions necessary to maintain a high standard of wound care in the wards.

The Medical Research Council Memorandum Number 6 gives a splendid account of the measures required, and a copy of this small but important pamphlet should not only be in every children's surgical ward, but should also be used continuously as a yard stick to measure up the existing standard of surgical hygiene within the ward.

I should like to lay emphasis on one or two important factors involved in this question.

The danger of nasal infection in post-operative palate cases, with resulting involvement of the suture lines, is a very real one. This, in my experience, never occurs in private hospitals, but occurs from time to time in public hospitals. Three possible causes are carelessness by the staff in the use of masks, overcrowding, and visits from relatives and ambulatory patients in the ward who have a respiratory tract infection.

Burns in children are liable to cross infection, whatever method of treatment is used, that interesting organism *Pseudomonas pyocyanea* being one of the troublesome contaminants; but this menace does not seem so difficult to combat with the exposure method as with pressure dressings (Colebrook, Duncan and Ross).

There is very great danger that the prophylactic use of antibiotics in surgical cases will lead to a general lowering of the standard of precautionary measures; the use of prophylactic antibiotics is indeed a two-edged sword.

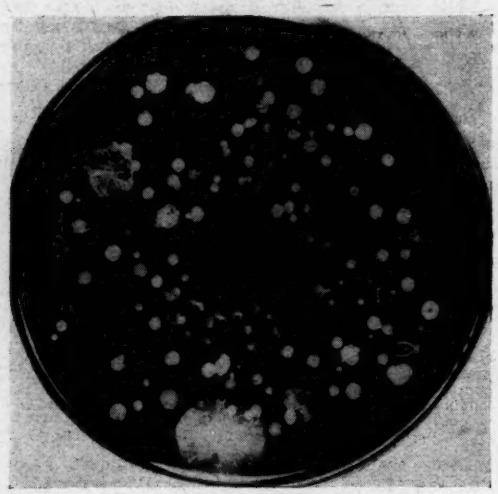

FIGURE VIA.

Slit sampler readings of duct air showing the efficacy of adequate ventilation and filtration in a Red Cross blood bank filling room. Fan off; filter off; number of colonies 170.

The work of Rountree and Thomson showed that the nasal carrier rate of penicillin-resistant *Staphylococcus pyogenes* was eight times as great in hospital staff members as in an unselected group of blood donors. Also in 56 cases of infection of clean surgical wounds with staphylococci they found that in 80% of the cases the incriminated organisms were penicillin-resistant. I remember that more than three years ago during routine investigations on three concurrent cases of burns infected with staphylococci I found that the organisms were penicillin-resistant in each case and all three patients had received prophylactic penicillin therapy.

Rountree and Thomson also showed that, with the increasing use of streptomycin, there was a significant increase in streptomycin-resistant strains of staphylococci in cross infected patients and in staff carriers.

It appears that up to the present antibiotic-resistant strains of hæmolytic streptococci are still rare (Rountree), though how long this favourable state of affairs will continue no one can say. It is my firm opinion that the prophylactic use of antibiotics should not be indiscriminate: they should be reserved for those cases in which infection would have very serious consequences, and their correct use will be influenced from time to time by further experimental work on the behaviour of the common ward organisms in response to exposure to the individual antibiotics.

The menace of air-borne infection should never be lost sight of, although its relative importance remains undetermined. In Australia, contamination of ward air with staphylococci is a much greater menace than contamination

with streptococci. Singer has found that this holds good also in other tropical and subtropical countries. Blankets are a potent danger and should always be handled gently to minimize contamination of the surrounding air. In hot climates, of course, their limited use makes them less dangerous. There is no doubt, however, that blankets

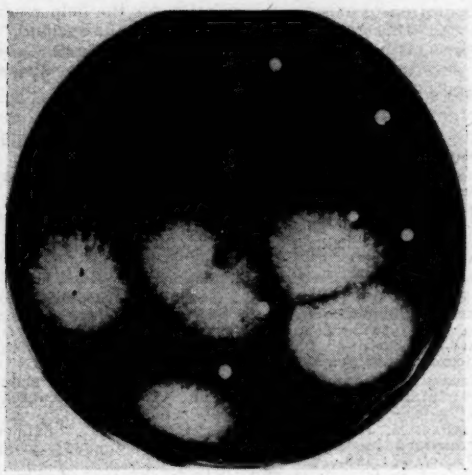

FIGURE VIb.

Slit sampler readings of duct air showing the efficacy of adequate ventilation and filtration in a Red Cross blood bank filling room. Fan on; filter off; number of colonies 15.

always constitute a menace. They cannot be boiled, and staphylococci have been found to survive vigorous and prolonged washing of blankets in hot water. Further, a blanket known to be sterile can be contaminated by the bacterial flora of the blankets with which it is washed.

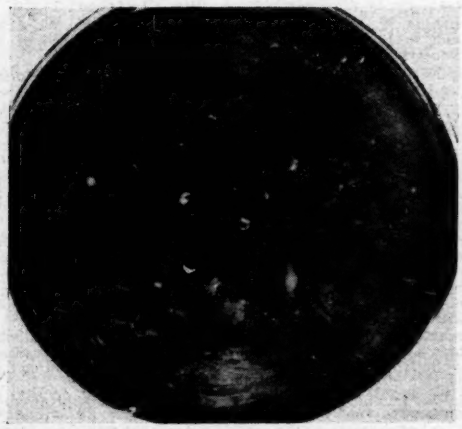

FIGURE VIc.

Slit sampler readings of duct air showing the efficacy of adequate ventilation and filtration in a Red Cross blood bank filling room. Fan on; filter on; number of colonies nil. (Note: The marks on the plate are photographic highlights.)

Rountree, and more recently Barnard and Leslie, have written interesting accounts of sterilization of blankets by oily impregnation with cetylpyridinium bromide, known commercially as "Fixanol C". Their work would seem to justify the adoption of this treatment for blankets, at least in special surgical wards and in the operating theatre.

I have had little experience in the oiling of floors as a measure to minimize air-borne infection, but Shaw has found "Precipitron Oil—Adhesive Type X" most effective on linoleum. Admittedly, oil cannot be used on certain other types of floors. Whether floors are oiled or not, they should be treated with an electric vacuum cleaner, and on no account should they be swept; preferably the vacuum cleaner should be connected to an air-tight exhaust outlet leading away from the ward.

The training of the ward staff, and indeed of the resident medical officers also, in the proper use of dressing trolleys and trays in the handling of wounds is of paramount importance. Whatever method of doing dressings is used, the vital requirement is to drive into the minds of all concerned the importance of the immutable barrier that must exist between "surgically clean" and "surgically dirty".

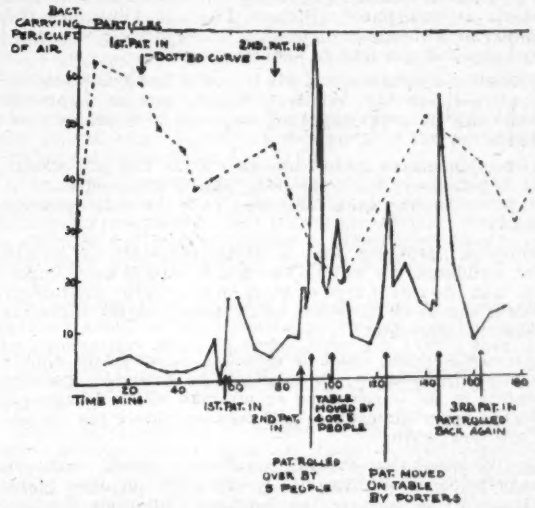

FIGURE VII.

Dotted curve shows exhaust ventilation system with contaminated air being sucked into the theatre from the anæsthetic room and the plaster room. Continuous curve shows same theatre after installation of an air conditioning plant providing filtered air at slightly positive pressure. The two sudden rises in this otherwise satisfactory curve show the result of forcible movement of a 17 stone man. (By courtesy of Her Majesty's Stationery Office, London, and R. R. Bourdillon, A. M. McFarlan and J. C. Thomas, "Studies in Air Hygiene", 1948.)

The use of masks, which is of extreme importance when dressings are being done in the wards, is discussed briefly later.

Cross Infection in the Operating Theatre.

The measures to prevent cross infection in operating theatres have probably been given more prominence than those for any other part of the hospital. Sterilization, setting-up and theatre behaviour are all well standardized procedures which it would be out of place to discuss in detail here, but there are a few factors of great importance on which I feel sufficient stress is not laid.

The very fact that methods have become standardized has led to a lessening of interest on the part of the doctor in the vital details of sterilization, and I very much doubt if many young graduates could write, impromptu, an intelligent essay on operating theatre asepsis and all it entails.

Reports of deaths following tetanus and gas gangrene contracted in the operating theatre (Sevitt and Robinson *et alii*) make one realize how important it is to ensure that autoclaving sterilization is effective—that is to say, that it ensures exposure of all articles sterilized by steam

to a temperature of 120° C. for ten minutes at least. This can be achieved in a good autoclave with a twenty pounds pressure and a ten inch vacuum, provided the bundles are correctly packed.

Figure II depicts the variation in performance of some autoclaves recently tested by Johnson. He also showed that correct packing of bundles in the sterilizer, and limitation of their size and weight are of vital importance (Figure III).

In addition to constant thermometer checks, test capillary tubes should be placed from time to time in the middle of bundles at the bottom of a load. The almost white tubes contain benzoic acid and a small quantity of impregnated methylene blue; at 121° C. the benzoic acid melts and the contents of the tube become uniformly blue.

For dry sterilizing in hot air ovens similar tubes containing salicylic acid, which melts at 158° C., can be used.

The value of masks in reducing the number of air-borne bacteria is undisputed (Figures IVA and IVB), but the question of which type of mask is most suitable is a very vexed one and can lead to endless discussion.

Filtration masks made of five layers of fine gauze (eighty threads per inch) are relatively efficient and are improved by the use of a "Cellophane" inset if it is properly in position.

Deflection masks made from used X-ray film are reasonably satisfactory, but have been proved more effective if five layers of fine gauze are sewn over the side openings (Shaw).

However, provided that a mask conforms to certain fixed standards, the way in which it is used is more important than the actual type of mask that is worn; improperly used, it can be as dirty and as dangerous as the infamous pocket handkerchief.

Air-conditioning, which is essential in our State, should be carefully installed; there should be a slightly positive pressure in the theatre, and an air inlet which diffuses air into the upper atmosphere and does not direct the current of air downwards.

A "Westinghouse Precipitron Filter", which works on the principle of electrostatic precipitation on oiled plates (Figure V) is used in the Brisbane Children's Hospital air-conditioning plant, and experiments carried out in the Red Cross Blood Bank in Brisbane have shown the effectiveness of adequate ventilation and air filtration (Figures VIA, VIB and VIC).

It is very easy to be lulled into a false sense of security by the adoption of ritualistic precautionary measures without due regard to their practical significance; the use of overboots is a case in point. They are, of course, of real value only if they are worn strictly within a prescribed area and never beyond it, and if at the same time no one walks into the prescribed area without overboots.

The ultra-violet lamp is another device which should be used only if it is installed and used skilfully with due regard to its limitations.

In order to keep a sense of proportion it must be realized that, whatever precautionary measures are taken, the human factor still plays a very large part in the hazards of the operating theatre.

Figure VII shows the danger inherent in exhaust ventilation systems and the tremendous increase in air-borne bacteria when there is considerable vigorous movement in the theatre.

Conclusion.

In the time available it has been possible only to paint in sketchy fashion some of the highlights on the vast canvas of this cross infection picture, but I have endeavoured to bring to notice, as a pædiatric surgeon, our responsibility in this great problem.

In conclusion, let us not forget that the menace of cross infection is always with us and is responsible each year not only for delayed and imperfect recoveries, but, in a percentage of cases, for actual deaths in what would otherwise have been satisfactory surgical cures, and let this awareness make us responsible for seeing, as far as it lies in our power, that we ourselves and all who work with us are unceasing in our efforts to minimize this ever-present danger.

Acknowledgements.

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THE CONTRIBUTION OF THE LABORATORY TO THE PROBLEM OF HOSPITAL INFECTION.[1]

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THE subject of cross infection is of such wide scope as to demand restriction of the remarks made in this contribution to the problem produced in a pædiatric hospital.

Some important general principles should first be revised concerning the types of spread of pathogenic organisms: (*a*) intermediate vector transmission, unimportant for the most part in temperate climates; (*b*) liberation of the pathogen into the fæces, sputum or other emanation, and its subsequent conveyance; (*c*) direct bodily contact; (*d*) droplet infection. Burnet (1946), in his "Background of Infectious Diseases in Man", selects these methods of transfer in his approach to the problem of infectious disease as an example of pathogen-host relationship, and this evolutionary outlook on the problem should guide our approach to the eradication of disease transmission with more effective results than any other.

Pathogenic bacteria, if the concept of evolution is applied to them, have reduced themselves toward a parasitic existence requiring, as exemplified in the extreme by the viruses, products of living tissue itself for their survival.

A means of effective transfer from one susceptible host to another is therefore essential for their survival, and, of course, eradication of such means equally a prerequisite of their destruction.

Cross infection is manifested clinically as respiratory, gastro-intestinal, wound, skin or mucous membrane infection and is the outcome of the acquisition of a pathogenic organism different from the one present on the patient's admission to hospital.

It is stated in the Medical Research Council Memorandum Number 11, 1951, "The Control of Cross Infection in Hospital", that respiratory infection is the most prevalent type of cross infection in hospital.

Whilst it is possible to say from post-mortem records that respiratory tract infection is a prominent cause of death in infants, it is difficult to assert from experience in the Royal Children's Hospital, Melbourne, what proportion of infections were actually contracted in hospital. In a brief survey of one hundred cases seen at necropsy my colleague, Dr. Alan Williams, has assessed that of 36 patients with signs of pneumonia 20 developed pneumonia whilst in hospital.

The question is complicated by the problem of terminal respiratory tract infection from which may be recovered only organisms usually regarded as commensals. Much has been written of the approaching ubiquity of the antibiotic-resistant *Staphylococcus aureus* in the respiratory tract of institutional dwellers where penicillin and other drugs are widely used. So far this organism has done little more than cause a shift to another antibiotic, but during the past few months in the Royal Children's Hospital, Melbourne, eight subjects with fatal and astonishingly destructive bronchopneumonia, some with empyemata, have been examined at necropsy. These cases came from without, and speculation was rife as to what set of circumstances led to this run of fatal staphylococcal pneumonias.

One hypothesis, that the infants had been victims of influenza infection and had reacted unfavourably to the secondary invasion of the staphylococcus, held sway but was not substantiated by demonstration of virus or antibody to the virus in our laboratory.

Reference to the danger of antibiotic-resistant staphylococcal lung infection as a possible complication of influenza epidemics of the future has been made by Sir Macfarlane Burnet in a recent address at the Royal Melbourne Hospital.

It seems that these children were invaded by a staphylococcus which was penicillin-resistant, and the organism established itself before the admission to hospital of the patient. Whatever the resistance-reducing factor, be it common to all or different, reduced resistance exists in many of the children in a pædiatric hospital. Are some of our terminal pneumonias cross infections? The answer must be "yes", but perhaps more pertinently may we not ask, would our cross infection not be much more overt if it were not for antibiotics?

Our use of antibiotics in a way does blanket the morbidity of cross infection and may blur the perception of it as far as respiratory tract infection is concerned. This is doing some good, but is treating cross infection without necessarily preventing it.

The modest part played by streptococci in the general medical ward cross infection illustrates the advantage of an organism's retaining its sensitivity to penicillin. Rapid diagnosis by the laboratory, and subsequent adequate antibiotic therapy, eliminate the organism. The figures for two months' throat swabs in the Royal Children's Hospital, Melbourne, show a 48% recovery of β-hæmolytic streptococci, but most of these are from the casualty department and general clinic where the treatment of the patients is carried out successfully.

The fortunate gaps in the circuit of cross infection are occasionally filled by the less fortunate consequences of antibiotic treatment, so that thrush occasionally makes its appearance and fatal outbreaks of *Monilia albicans* infections have been reported in infants in maternity wards (*Brit. M. J.*, 1950).

Throughout the respiratory tract, whether from the auditory canal or the bronchioles, we are substantiating the observations made by Rountree *et alii* (1951) that the *Staphylococcus pyogenes* has become increasingly antibiotic-resistant, making the selection of drugs difficult and delayed.

About three-quarters of wound swabs in our hospital from surgical wards have in them a *Staphylococcus pyogenes*. In one series taken out over a fourteen months period (January, 1953, to March, 1954) out of 205 cases with a pathogenic organism 148 cases showed *Staphylococcus pyogenes*.

Pseudomonas pyocyanea, which we usually regard as a good index of cross infection, was found in only 5% of cases in this series. One would regard this as a definite indication that wound-to-wound transfer is less contributory to cross infection than is transfer from the nasopharynx of the attendant.

It would be pertinent for a surgeon from our own hospital to discuss the effects of hospital infection, touching perhaps on the subjects of burns, skin grafting and the delay if significant that hospital infection causes to discharge of the patient from hospital.

It is interesting to note that the number of salmonella or shigella infections turning up from the surgical wards is extremely small. This is due, no doubt, to the rarity of a patient with this kind of organism being admitted to a surgical bed.

Entero-Colitis.

The problem of a child suffering from a disease in which pathogenic organisms are excreted in the fæces contrasts unfavourably with the same problem in an adult.

More actual handling of the infected material is unavoidable even if the salvage of the soiled napkin is the only factor considered.

Other factors include the infection of the infant's environment by its own spread of fæcal contamination and the unwitting handling of objects by staff which are least considered likely to be contaminated.

A lively imagination is all that is necessary for a progressive count of the dangers in an entero-colitis ward, and

[1] Part of a symposium on "Cross Infection in the Hospital" held at the annual meeting of the Australian Pædiatric Association, Canberra, April 8 to 11, 1954.

it is to be remembered that, although the age incidence of shigella infection is highest in the two to three year range, that of salmonella is highest at about seven months, a most uncooperative time even of a healthy child's life in matters of hygiene.

No one denies that in the combating of cross infection in a pædiatric entero-colitis ward, the patient is written off as a cooperative unit and the whole onus of prevention is on the staff.

The importance of hospital design and ward appointments becomes apparent if facility of movement and reduction of fatigue are to enable the staff to carry out its onerous task.

In our hospital it is nearly seven years since cross infection has caused anything like critical trouble, when an outbreak of *Salmonella derby* enteritis pointed out all the hazards of an intramural outbreak from the significant victimization of weakly and premature infants to the rather frightening mortality.

Since then many improvements in patient handling, preparation and delivery of formulæ and disposal of contaminated squares have resulted in what seems to be a permanent reduction in the incidence of cross infection in the entero-colitis ward.

The contribution of the laboratory to this improvement has been that of one member of a large team including medical and nursing staff whose attention has been directed to the problem. It is easy to recollect the value of the lightning ward survey, when a suspicion of cross infection is present, to attempt to locate the spreader. Our attention may have been in the past too much directed to the patient culprit rather than the slightly ill ambulatory adult. Singer and Ludford (1953) have drawn attention to the danger of ambulatory carriers or staff with a mild attack of diarrhœa in a children's institution.

In the discussion of entero-colitis in infancy one must refer to the challenging problem of ætiology with the low rate of recovery of organisms.

In our laboratory there are in use orthodox modern techniques for the isolation of salmonella, shigella and pathogenic *Bacterium coli*, but we do not routinely pursue the staphylococcal and food poisoning group.

In a recent publication the value of phage typing in staphylococcal enteritis has been emphasized (Crowe, 1954) and it is our intention to pursue this aspect of entero-colitis in infancy in the near future. In a recent investigation my colleague, Miss Friday, working under a grant from the National Health and Medical Research Council, has shown that improvement of bacteriological scrutiny by the addition of serological testing yielded in a group of ninety-six cases almost a 20% improvement in specific diagnosis, raising the final pathogens isolated from 43% to 62% in this group of ninety-six.

This work is in the process of preparation for publication, and it emphasizes the insecurity of relaxing precautions simply because organisms have not been isolated.

It would be improper even in this modest excursion into some aspects of the problems of entero-colitis in infancy to omit the significant changes that have taken place in the treatment of the disease resulting from improvement in the diagnosis and correction of fluid and electrolyte imbalance.

It is always well to remember dramatic changes like this when assessing the results of other approaches to the elimination of the disease, whether they be by the use of drugs or other devices.

In conclusion, it would in my opinion be presumptuous to over-emphasize the place of the laboratory in the prevention of cross infection, but dangerous to cast loose this valuable member of the routine and research team.

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A B O BLOOD GROUPS AND FŒTAL ERYTHROBLASTOSIS.[1]

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THE discovery of the Rh factor soon led to the realization that Rh incompatibility between mother and fœtus can result in a maternal immunization expressing itself in the development of Rh antibodies and consequently in the damage of fœtal tissues by these antibodies, giving rise to the syndrome known as *erythroblastosis fœtalis* or hæmolytic disease of the newborn.

It was soon discovered that identical disease in the offspring could arise as a result of maternal sensitization to the A B O blood factors, in an A B O incompatible or heterospecific pregnancy. Thus the disease may arise as the result of an incompatible mating in respect of the A B O, Rh-Hr or other rarer factors in the blood. A compatible mating may be defined as one in which both parents belong to the same A B O or Rh groups, or in those in which the dominant A or B or Rh is present in the mother's blood. In an incompatible mating, giving rise to a heterospecific pregnancy, the dominant factors A or B, or both, or Rh are present in the father but lacking in the mother. Incompatible matings occurred in 35% of all marriages in a series in the United States of America, and in 22% of our own Melbourne series. A B O incompatibility is particularly interesting, as there are frequently in the maternal blood naturally occurring immune antibodies against A and B substance, which appear to be genetically determined, and, furthermore, a rise in the titre of these antibodies is known to occur following a number of stimuli, for example:

(i) Injections of TAB and horse serum *et cetera.*

(ii) Ingestion of certain foods—for example, meats and legumes which contain naturally occurring group-specific substance.

(iii) Infections due to certain viruses and bacteria.

(iv) Spectacular rises due to fœtal stimulation in heterospecific pregnancies. The last is not necessarily the beginning of immunization, but may be a more vigorous continuation of a process of long duration. However, if immune (or incomplete) antibodies corresponding in A B O specificity to the child's blood group are present in sufficently high titre, they may cause damage as severe as that caused by Rh antibodies.

The frequency and severity of disease in the offspring of heterospecific pregnancy have not previously been reported, because the ante-natal detection of maternal isoimmunization to these factors has presented a much more difficult problem than that of sensitization to the Rh factor. In fact, Levine (1953) states:

> At present the occurrence of A-B hæmolytic disease in pregnancy cannot be predicted even in a mother who previously gave birth to an infant with this disorder. The obstetrician is never aware of trouble before the birth of the infant. The mother is usually Rh positive and there is a considerable delay (24 to 48 hours, or longer) before the cause of the jaundice is investigated. The diagnosis is not as simple as in Rh incompatibility.

[1] Read at the annual meeting of the Australian Pædiatric Association, Canberra, April 8 to 11, 1954.

At the Queen Victoria Memorial Hospital, Melbourne, after a number of poor results and a few fatalities due to late recognition of this condition, Dr. Rachel Jakobowicz suggested a method for detection of A B O sensitization in the ante-natal period, and thus after twelve months of systematic testing, extending from the end of February, 1953, until the end of February, 1954, we are able to present to you some figures which answer some of the unknown features of this condition.

A sample of blood is collected from each woman attending the hospital on her first ante-natal supervision visit. The A B O and Rh status of these are determined and investigation is made for the presence of Rh immune antibodies. Dr. Jakobowicz then tests each serum for the presence of isohæmolysins, which, if present, indicate sensitization of some variety. All sera demonstrating isohæmolysins in high degree are then tested for the presence of anti-A or anti-B immune antibodies, and if they are detected, monthly specimens of blood from these women are submitted to determine whether there is any alteration in the titre which would suggest fœtal stimulation. In this way we have been able in many cases to forecast with accuracy the infant's blood group. A rise in titre, while not absolutely indicative of disease in the offspring, has been found to be a useful pointer which makes us alert to the possibility of an affected infant and allows us to be prepared.

The results during the twelve months' period mentioned are as follows.

The total number of women tested was 4511; of these, 3820 were delivered before March 31, 1954. Approximately 1000 (22%) of this total of pregnancies were heterospecific. One hundred and twenty-five women exhibited a high degree of isohæmolysins in their serum—that is, 3%. Of these, 111 women have been delivered. The total number of affected infants is nine (8·1%); four of these were first-born infants; five were severely affected and required exchange transfusion (one of these had a positive direct response to the Coombs test). Four were mildly affected and were treated conservatively. All recovered without sequelæ. A further 380 women developed immune antibodies at or after delivery. The percentage sensitization of women to A B O factors was 11.

Of 111 delivered mothers with high antenatal A or B antibodies, 44 group O mothers had A offspring; four were severely affected (received exchange transfusion), three were mildly affected (jaundice), one was anæmic and had a weak positive direct response to the Coombs test. Fifty-five mothers of group O had unaffected group O offspring. Eleven mothers of group O had group B offspring; one was severely affected (received exchange transfusion), one was mildly affected (jaundice). One group B mother had an unaffected group A offspring. Of the four first-born affected offspring, one was severely affected (received exchange transfusion), three were mildly affected (jaundice).

In order to compare this incidence with that of Rh sensitization in the same group of women we have the following figures. The total number of women tested was 4511; of these, 3820 were delivered before March 31, 1954. Approximately 721 women were Rh negative (16%). An estimated 433 offspring were Rh positive (60%). Forty-three Rh-negative women developed antibodies antenatally (five of these developed the antibodies at delivery); this was 1% of the total. The percentage of Rh-negative women sensitized was 6·1.

The total number of affected infants was 14 (32·5%). The total deaths of affected infants was four (9·3%). Two of the four died from *hydrops fœtalis*, and two died during exchange transfusion. Ten infants were severely affected (received exchange transfusion). Two infants were mildly affected (treated conservatively). Thirteen pregnancies have not been terminated to date. Five infants were Rh negative and thus unaffected. Two pregnancies ended in abortion. All affected infants had positive direct responses to the Coombs test on cord blood. All other pregnancies resulted in normal infants.

From these figures the chance of sensitization to the Rh factor (6·1%) is approximately half the chance of sensitization to A B O factors (11%). However, the chance of having an affected infant is almost five times greater in the group sensitized to the Rh factor than in the group sensitized to A B O factors. In this series 82% of the infants affected by Rh antibodies were moderately or severely affected, but approximately 50% of infants affected by A or B antibodies were severely affected; and in this series there was no instance of *hydrops fœtalis* from this cause. Clinically hæmolytic disease due to A B O sensitization differs from that due to Rh sensitization in several important respects (see Table I).

TABLE I.

Differentiating Features of Hæmolytic Diseases.[1]

Feature.	Due to Incompatibility of	
	Rh.	A-B.
Frequency (*per centum*) of incompatible matings	13.	35.
First-born affected	Rare.	About 50%.
Influence of previous transfusion on affected first-born	Important.	None.
Secretor status of infant	Non-secretor of Rh.	Secretor.
Factor in tissue cell	Absent.	Present.
Duration of hæmolytic process	Long.	Usually short.
Direct Coombs test on cord or peripheral blood	Strong.	Weak or absent.
Red blood cells on blood film	Macrocytosis.	Microspherocytosis.
Osmotic fragility	Usually normal.	Increased.
Hæmolysin in maternal serum for infant's blood	Absent.	Present.
Clinical condition at birth	Jaundice early.	Jaundice early.
	Marked anæmia.	Anæmia not so marked.
	Hepatosplenomegaly.	Spleen not always enlarged.
	Liability to kernicterus.	? Increased liability to kernicterus.

[1] Modified from Levine *et alii*, "Advances in Pediatrics", Volume 6.

As some women have an extremely high A or B antibody titre, even before their first pregnancy, clinical disease occurs in 50% of first pregnancies, whereas disease in the first-born is rare in Rh sensitization.

Anæmia.

In severe cases of Rh disease there is a macrocytic anæmia, the red corpuscles are unusually large, and there is polychromatophilia, with large numbers of normoblasts or erythroblasts showing all phases of maturation.

In cases of A B O disease the anæmia is not so profound, the lowest cord blood hæmoglobin level in this series being 75% (11·25 grammes *per centum*), in contradistinction to levels of 50% (7·5 grammes *per centum*) or less in several of the Rh cases.

The red blood cells exhibit microspherocytosis and increased fragility; there may be a reticulocytosis and normoblastæmia. Splenomegaly is not so common in A B O disease; in fact, the brunt of the disease seems to fall on the tissues rather than on the red blood cells. It seems probable that kernicterus and damage to developing fœtal organs is more common than anæmia in this disease. It has been proved that immune A and B antibodies are of small enough molecular volume to pass the placental barrier and reach the fœtal circulation; but the infant's red cells are in part protected from damage by the presence of group-specific receptors which neutralize the antagonistic maternal antibody. Of the babies affected with A B O disease, 100% are secretors—that is, they have water-soluble group-specific receptor substances in their tissues and body fluids. This secretor state has a double function: it is the cause of the maternal immunization, the antigen being not the fœtal red cell but these placenta-permeable group-specific substances, and at the same time these substances act as a partial protection to the red cells through neutralization of the antagonistic maternal antibodies which they have caused to increase.

This probably accounts for the fact that the direct response to the Coombs test on the cord blood is usually negative in A B O disease, whilst it is a sensitive indicator of immunization in Rh disease.

Finally, the most important single clinical sign in both conditions is the early development of jaundice, and the most useful form of treatment so far devised for the moderately or severely affected infants is exchange transfusion.

TENSION EMPHYSEMA: A SURGICAL EMERGENCY IN INFANTS.[1]

By T. Y. NELSON and DOUGLAS REYE,
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THE desire to report the following cases was prompted by the fact that the first of the series presented as an emergency; its true nature was not recognized and in the study of the subsequent post-mortem material a condition was revealed which should be amenable to surgery.

The condition is one of lobar emphysema which usually affects the upper lobe of the right or left lung or the middle lobe of the right lung. The affected lobe becomes grossly over-distended, causing collapse of the remaining lobes and a considerable degree of mediastinal displacement. This may be an acute emergency which is rapidly fatal if unrelieved, or the history may be that of a child who wheezes or has some degree of dyspnœa which persists to a varying degree for months. The development of an acute respiratory infection with exacerbation of the symptoms prompts investigation in hospital when X-ray examination should disclose the true state of affairs.

The cause of this condition appears to be a faulty development of the bronchus supplying the affected lobe, in which there is a deficiency of cartilage allowing the bronchus to collapse. This produces a flap-valve mechanism by which air is allowed into the affected lobe but cannot be expelled from it.

Reports of Cases.

Case I.

A male infant (C.N.) was born at the Royal Hospital for Women, Sydney, on June 8, 1953, after an easy instrumental delivery. The baby was unusually asphyxiated for such an easy birth, and resuscitation took possibly ten minutes. The resident medical officer noted that the apex beat was on the right side and this observation prompted early X-ray examination, which revealed marked mediastinal shift and appearances in the left lung field which at the time were thought to indicate a tension cyst (Figure I).

The baby was transferred to the Royal Alexandra Hospital, Sydney, where in addition to the cyanosis and dyspnœa marked recession of the chest was seen. A needle introduced into the chest and connected to an underwater drain released only a few cubic centimetres of air and gave little relief. The baby died in a few hours.

At the autopsy the thoracic viscera were removed intact and it was seen that the upper lobe of the left lung was grossly emphysematous, with collapse of all the other lobes. When the removed viscera were viewed from the posterior aspect the artery to the lower lobe of the left lung was found to cross the left upper lobe bronchus, and, though in a normal anatomical position, it appeared to flatten the bronchus, which was abnormally soft and compressible.

Serial sections were examined from a block which included the left main bronchus together with the bronchi to the upper and lower lobes, and their second division branches within the lung. The left faulty upper lobe bronchus could be clearly traced in these sections. From its point of origin in the left main bronchus it had an oval outline due to collapse of its hypoplastic walls, which were entirely devoid of any cartilaginous support. As it entered the lung substance and divided, however, it altered suddenly. The lumen became rounded and cartilage bars appeared, the only departure from the normal at the more distal level being a small deficiency in cartilage at two diametrically opposed points in the circumference. As was expected, the structure of the left main bronchus and the bronchus to the lower lobe of the left lung were entirely normal.

These findings were accepted as those of an unusual case until on June 24, 1953, a child (C.D.) of seventeen months was subjected to operation for what had been diagnosed as a cyst of the upper lobe of the right lung and was found on examination of the specimen to have a similar condition.

Case II.

A child was first admitted under the care of Dr. S. E. L. Stening at the age of nine months, with a history of "panting", persisting since the age of two months. She recovered from an attack of bronchitis at the age of five months, but the dyspnœa was worse after this event and her general progress was poor. Radiographs taken at this stage showed herniation of the right lung through the anterior mediastinum, and it was thought there were at least two large cysts on the right side (Figure II). Her condition improved and she was kept under observation for some months, but as no change occurred in the radiological picture the decision was made to perform lobectomy.

At operation the upper lobe of the right lung was grossly distended and did not collapse when the thorax was opened. The upper lobe was removed, and convalescence was satisfactory after a tension pneumothorax had been relieved on the day following operation.

Examination of the specimen showed greatly over-distended right upper lobe with the characteristic appearances of a diffuse emphysematous enlargement. Part of the right upper lobe bronchus was visible projecting slightly at the hilum, and this bronchus was soft and readily compressible. A cross section of the entire bronchus is shown in Figure III. The mural hypoplasia is clearly evident, as is the almost total absence of any cartilagenous support.

With these two cases in mind treatment by immediate lobectomy was planned if such a case presented as an emergency. The difficulty was to decide the degree of emergency, as the second patient had survived an acute respiratory infection and had recovered well after a deliberate lobectomy eight months later.

There was not long to wait, for the third case appeared on June 27, 1953, again under the care of Dr. Stening.

Case III.

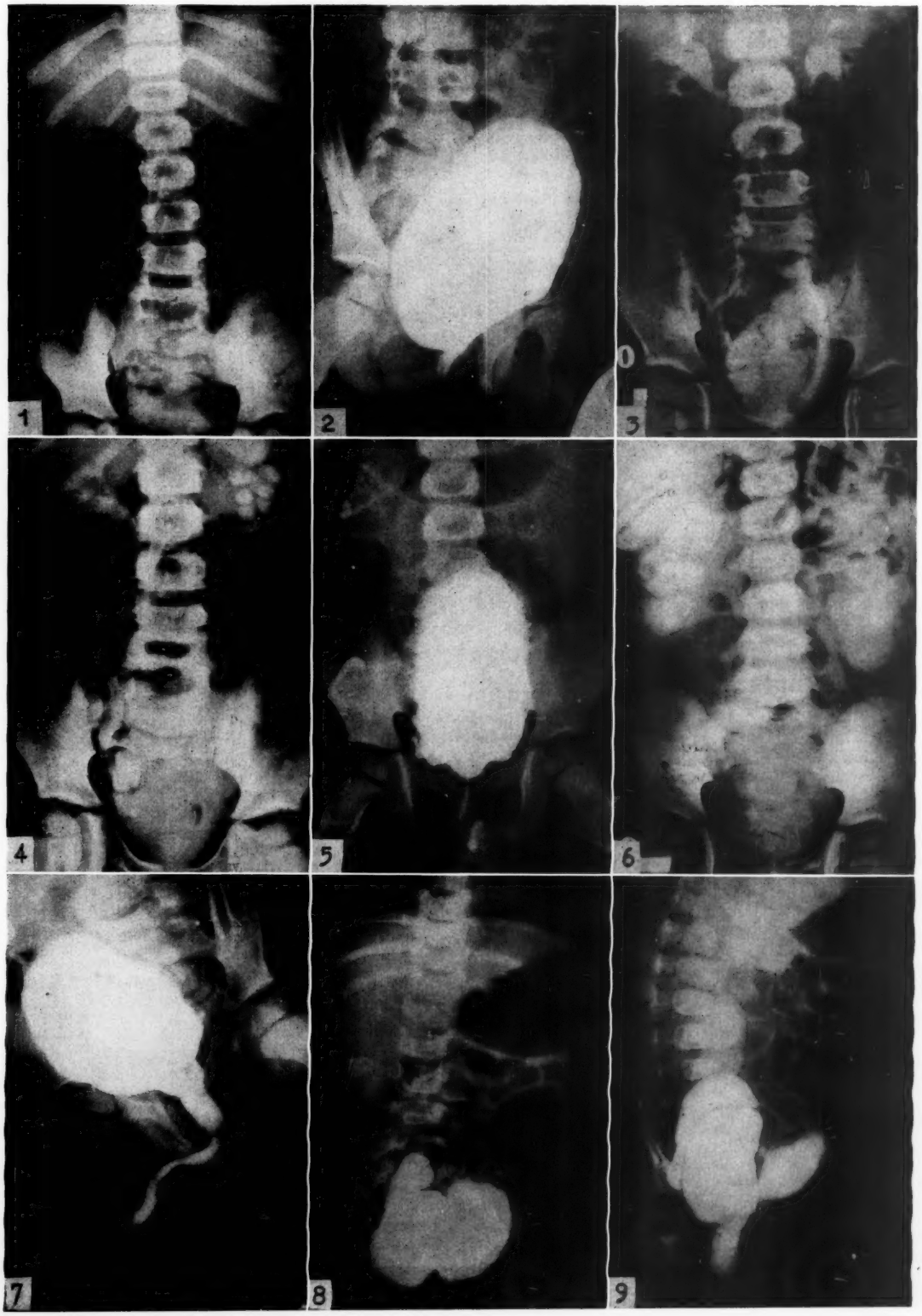
A five weeks old baby (B.H.) had been well for the first three weeks of her life and then developed an upper respiratory tract infection. One week later dyspnœa and respiratory spasm were noted and the baby improved after treatment with penicillin and "Aureomycin". She was admitted to hospital after a period of three days in which she had become much worse with marked cyanosis and dyspnœa and the passage of loose green stools. After her admission to hospital a laryngeal swab revealed the presence of *Staphylococcus aureus*, and her condition steadily deteriorated. Urgent lobectomy was advised and carried out on July 2, 1953. The chest wall was infiltrated with local anæsthetic and the thoracotomy carried to the stage where the rib was removed, but the pleura was not incised. At this stage the anæsthetist intubated the child, the pleura was incised and the emphysematous upper lobe delivered. The pressure was relieved and the atelectatic lower lobe could be inflated, and there was an immediate improvement in the child's condition. Lobectomy was then performed without difficulty, and the child was returned to the ward in a reasonably satisfactory state. Her condition, however, did not improve and she died on the night of operation.

An emergency operation of this type performed during the course of an acute staphylococcal infection can be justified only by the deterioration which was obviously occurring, and raises the question whether this condition would not be better treated as soon as it is recognized and before such an infection makes successful lobectomy hopeless.

The emphysematous upper lobe of the left lung removed at operation resembled the lobes in the two previous cases. The upper lobe bronchus could not be found in the specimen, but sections through the hilar region contained bronchi after division, and these bronchi were incompletely surrounded by cartilage, though not entirely devoid of it. In the subsequent autopsy material the left upper lobe bronchus was found and sectioned, and shown to have an inadequate development, while the bronchi to all other lobes were entirely normal. An additional feature in this case was the presence of an

[1] Read at the annual meeting of the Australian Paediatric Association, Canberra, April 8 to 11, 1954.

ILLUSTRATIONS TO THE ARTICLE BY EDWARD S. STUCKEY.



ILLUSTRATIONS TO THE ARTICLE BY T. Y. NELSON AND DOUGLAS REYE.

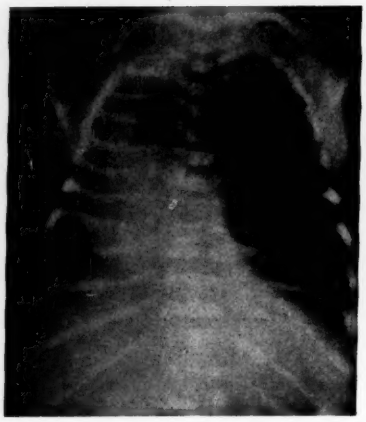


FIGURE I.

Shows emphysema of upper lobe of the left lung with herniation across the midline and mediastinal displacement. Lung markings can be seen in the emphysematous lobe.

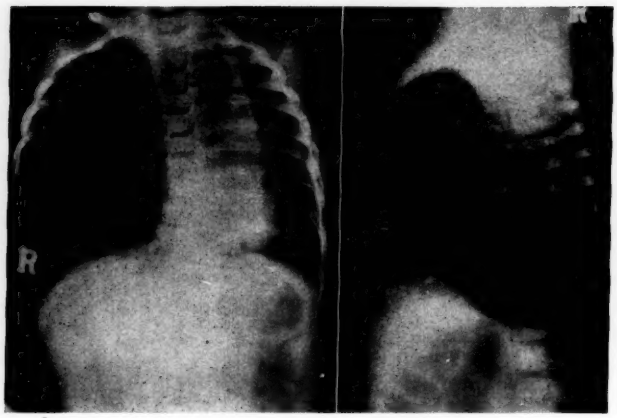


FIGURE II.

Shows herniation of right lung through the anterior mediastinum. This condition persisted without change for eight months.

FIGURE III.

Showing the typical maldevelopment of the bronchus. Mural hypoplasia is evident and only one small piece of cartilage can be seen.

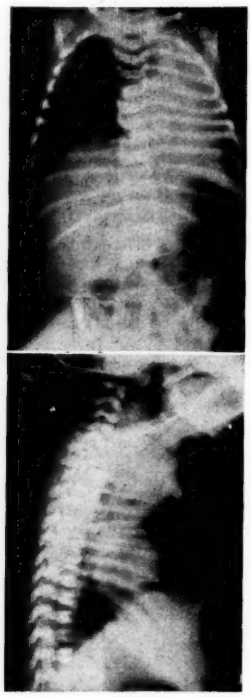


FIGURE IV.

Tension pneumothorax on right side with collapse of lung and extreme displacement of the mediastinum.

acute inflammatory exudate in the lumen of the abnormal and the normal bronchi, with extensive lobular pneumonia of all lobes.

Case IV.

During the same month Dr. E. S. Stuckey had a child under observation, a baby, aged three months (M.T.), suffering from an abnormal condition of the right lung with displacement of the heart to the left. During the investigation of this infant bronchoscopy was performed, and considerable narrowing of the right main bronchus was found which increased in calibre after suction. After this procedure the child's condition deteriorated, the heart and mediastinum were displaced even further to the left, and the right lung was in a state of extreme emphysema. Two days later the child had a temperature of 103° F., with marked chest recession, and a laryngeal swab grew pneumococci and *Micrococcus catarrhalis* insensitive to penicillin. This febrile episode subsided after treatment with "Aureomycin" and ten days later successful removal of the right upper lobe was carried out. At operation Dr. Stuckey found the right middle and lower lobes collapsed, the upper lobe bronchus was narrow and thin-walled, and the upper lobe was grossly emphysematous.

There were in this case no essential differences from those already described—that is, abnormal development of the main bronchus to the lobe—though it is worthy of note that some sections taken through other parts of the lobe showed an inadequate mural development while others showed normally developed bronchi.

Case V.

At present under observation is a child (C.J.) seen first in September, 1953, with a history of persistent wheeze. At the age of three months the child was thought to have pertussis, but recovered, and at the age of four months X-ray examination revealed emphysema of the left side, with mediastinal displacement to the right. The child's condition remains satisfactory, and he will be subjected to lobectomy at an early date.[1]

Discussion.

This condition is well known in pædiatric literature and has been recognized for many years; in fact in the records of this hospital is to be found a similar case which came to autopsy fifteen years ago. An added stimulus has been given to the subject by the advances that have taken place in thoracic surgery making early and successful treatment practicable.

The earlier isolated case reports of infants suffering from obstructive emphysema showed that the basis of the condition was usually absence of the normal cartilage rings in the affected bronchus. A flap of bronchial mucosa was thought to be the cause in two cases of a series reported by Robertson and James (1951).

Fischer, Potts and Holinger (1952) describe a series of five cases in infants for which lobectomy was done. In two cases cartilaginous rings were deficient and in two others the *ductus arteriosus* overlay the left main bronchus and was attached low down on the pulmonary artery. This was thought to be the obstructing mechanism, but in spite of this lobectomy was performed after the ductus had been divided. In one case there was found to be an associated bronchial abnormality, while no further abnormality was found in the other.

In a number of reported cases no abnormality of bronchial structure has been found.

Recently, Sloan (1953) has reported a further series of four infants on whom lobectomy was performed. He states that careful examination of the lobes removed revealed no specific bronchial obstruction, and attributes the condition to an abnormal flaccidity of the bronchial walls. This concept of bronchomalacia (Engel, 1947) is not, however, supported by sections of the bronchi, but was suggested by the findings at bronchoscopy that the bronchial walls approximated during expiration.

His cases differ from other reports in that the patients did not completely recover following operation. Each infant seemed prone to develop respiratory infections, and two were admitted to hospital several times with wheezing and dyspnœa. Later examination of two infants showed emphysema in other areas of the lung fields.

In the present series a definite abnormality of the bronchus was found in each case. Two infants followed the usual pattern of respiratory difficulty beginning early in infancy, and in two this became an emergency resulting in rapid death in the first case and a fatal termination following lobectomy in the other owing to a staphylococcal infection. This raises the question of urgency in dealing with the condition.

It does appear that there may be extreme urgency in the neo-natal period extending perhaps over some of the earlier months of infancy. In the later cases, if the child survives he can apparently carry on for a long period without symptoms. No report has been found in the literature of a patient having been treated conservatively over an indefinite period, and even Caffey (1950), who has made a plea for the conservative treatment of many lung cysts, recognizes that this condition is different and describes two cases recognized before the age of six months in which lobectomy was performed.

The diagnosis has presented difficulty in many of the reported cases, the usual mistake being to confuse the condition with a tension cyst, as was done in Case I of this series. Careful examination of the X-ray films should show the presence of lung markings through the affected area, which are the distinguishing feature. A tension pneumothorax will produce the same clinical picture in the neo-natal period, but the radiological appearance is distinct, as can be seen from Figure IV. In this case extreme dyspnœa was noted soon after birth and the child died in a few hours. There is marked mediastinal shift with evidence of air in the right pleural cavity, and the outline of the collapsed lung is well seen.

There can be little doubt that many of these cases are associated with a developmental defect of part of the bronchial tree. This may be of very limited extent, as the examination in Case I showed. It might be possible to miss such an area in the examination of an operative specimen if the bronchus was sectioned just distal to the affected segment. Further, the examination in Case IV suggested that the defect may be zonal in distribution, and this would explain the occurrence in a segment of a lobe (as seen in one of Shaw's cases) if the pores of Kohn were obliterated by an inflammatory condition.

Summary.

1. Four cases of emphysema of lobar distribution are recorded occurring in infants.

2. Two of these presented as emergencies and both patients died, one after lobectomy. The other two patients were cured by lobectomy.

3. In all four the cause was considered to be absence of bronchial cartilage in the bronchus of the affected lobe.

Acknowledgements.

It is a pleasure to acknowledge the assistance given by Dr. G. Sylvester in the interpretation of the radiographs, and by Dr. A. Distin Morgan in the difficult task of anæsthetizing these infants. The inclusion of Dr. E. S. Stuckey's case adds considerably to the value of the report.

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[1] This child has been operated on and was found to have the typical deformity of the left upper lobe bronchus. A further patient who has also been subjected to operation had the deformity in the right upper lobe bronchus. This makes six cases in all.

APPENDICEAL COLIC.[1]

By ROBERT SOUTHBY,
Melbourne.

THE problem of recurring abdominal pain in children—"belly ache" or "tummy ache" as they describe it—is an ever-present and very practical one. I am not referring to any of the acute inflammatory or obstructive conditions which present as surgical emergencies, but rather to the attacks of intermittent pain of short duration, recurring at varying intervals over a period of months or years.

Careful inquiry and clinical examination will disclose from time to time such conditions as ileo-caecal adenitis, infection of inguinal or deep iliac glands, arthritis of the hip joint, a spinal lesion, a tuberculous abdominal infection, recurrent intussusception, or even a rheumatic infection culminating in carditis. Having excluded such definite entities as these, we are still left with a group of children suffering from recurrent abdominal pain in whom the pain may be accompanied by anorexia and slight nausea, but rarely by vomiting; there may be no rise of temperature and only a slightly hurried pulse rate, and no disturbance of bowel or bladder activity.

The pain is definitely colicky in character and may last only for seconds or minutes at a time. While it lasts it is quite severe and "doubles him up" or "makes him cry", but as soon as it is over he is back at his games again. He may have a number of attacks in one day, may be disturbed at intervals through the night, or he may not have had a day quite free of pain for several months.

Occasionally there may be periods of a week or more, even up to a month or two, without any discomfort whatever.

General examination does not reveal any evident sign of organic disease and there are no localizing signs of abdominal inflammation, such as tenderness, muscular rigidity, or hyperæsthesia. This group of children provides a definite clinical entity which I term recurrent appendiceal colic, and the basis for the pain is mechanical rather than inflammatory, as is the case in recurrent appendicitis.

At this stage I should like to quote the clinical histories of a few of the children suffering from this condition.

1. B.M., a boy, aged five years, had recurring attacks of colicky abdominal pain referred mainly to the navel, and unaccompanied by vomiting. They persisted intermittently for over twelve months. There was some frequency of micturition and dysuria with one attack.

2. T.M., a girl, aged nine years, had had pain beside the navel for the previous three months. The pain was colicky and intermittent. The patient was nauseated, but no vomiting was present. She had some pain almost every day, but it did not disturb her at night.

3. U.S., a girl, aged seven years, awakened six weeks previously with rather severe pain in the left side of the "chest and tummy". She was seen by her local doctor and the mother was reassured that the condition was not appendicitis but merely a mild "indigestion". The pain persisted and was more severe, and on this occasion she was seen by another local doctor, who regarded the condition as a mild colic of some type. The pain persisted, and the mother's observation at this stage was to the effect that "pain does not continue unless there is some reason for it".

4. B.S., a girl, aged thirteen years, had had several attacks of abdominal pain in the previous eight months. The pain was persistent at first and then became colicky, but was not accompanied by vomiting and was independent of menstruation, which was quite regular and free from sickness or pain of any description.

Careful routine clinical examination of each of these children failed to reveal any evident sign of organic disease which could have given rise to pain of this character.

With our suspicion aroused as to the possibility of appendiceal colic, I feel that the radiologist can help us considerably in the elucidation of the problem.

During the opaque meal examination in normal health the appendix will usually fill with barium, thereby demonstrating its size and position, and it will generally empty as the rest of the bowel empties with the passage of the meal.

With some of these children the X-ray findings are those of a long and tortuous or kinked appendix, with perhaps areas of varying translucency consistent with the presence of fæcoliths, or possibly only a tiny amount of barium showing within the lumen; furthermore, this type of appendix generally does not empty. Such a report I regard as "positive" evidence in confirmation of our clinical suspicions.

With others the radiologist announces that he is unable to visualize the appendix, sometimes after several attempts both with screen and film. He usually adds a rider that there is no tenderness over the ileo-caecal region, which also does not appear to be fixed, and offers the conclusion that there is therefore no radiological evidence of pathological change in the appendix.

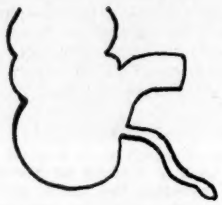

FIGURE I.
Normal appendiceal pattern.

I quite agree that the absence of tenderness would help to exclude a condition of inflammation, but on the other hand I have learned from experience that this so-called "negative report" is really very valuable in confirming the suspicion of appendiceal colic.

In almost every instance with such findings the appendix is found by the surgeon at operation to be either sharply kinked back at the base, or twisted upon itself, or firmly adherent to the caecal wall, or so narrowed in the lumen

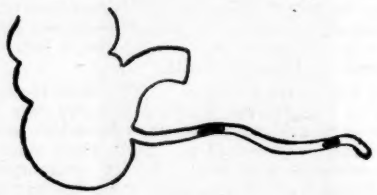

FIGURE II.
Unusually long with fæcoliths.

or completely blocked that it is impossible for barium to enter the appendix from the gut, and it is equally impossible for mucus or other content of the appendix to pass into the lumen of the gut. This stenosis or actual atresia is, I am sure, a congenital lesion in such children.

The varying types of shadow reported in the opaque meal examinations are represented in the accompanying diagrams, which are self-explanatory (see Figures I to VI).

Let us now revert for a few moments to the four case histories previously quoted and compare the opaque meal reports and the findings at operation. They are as follows.

1. B.M.—"The appendix was not visualized during the examination, but there was no tenderness on palpation."

"No X-ray evidence of appendiceal inflammation at the time of our examination. This, of course, does not exclude M.'s pain as being due to an appendiceal colic, as it will be noted that the appendix did not fill with the barium."

[1] Read at the annual meeting of the Australian Pædiatric Association, Canberra, April 8 to 11, 1954.

"The appendix was sharply kinked a short distance from the base by a peritoneal fold containing a blood vessel, and distal to the kink the appendix was dilated."

2. T.M.—"At twenty-four hours the appendix was very incompletely filled, and in spite of further barium and examination at forty-eight hours no further filling of the appendix took place."

"The appendix was a long one, adherent in its proximal half to the caecal wall and kinked at its centre and with a fæcolith in the distal half."

3. U.S.—"The appendix presented a segmented appearance, and the portion outlined was moderately mobile, but it is doubtful if it was completely filled to the tip."

FIGURE III.
Kinked.

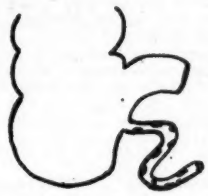

FIGURE IV.
Tortuous and mottled.

"The appendix was moderately long, tortuous, adherent at the tip and contained several fæcoliths."

4. B.S.—"The caecum was deep in the pelvis, and at no time was the appendix visualized."

"Gross ptosis of the caecum—a fairly long appendix with the lumen completely occluded at the base."

It will now be evident, I am sure, why I maintain that the pain in these attacks is mechanical in origin and therefore described as recurrent appendiceal colic.

I should like to thank all those of my surgical colleagues who have listened to my persistent appeals to have these children operated upon, and who, having acquiesced, have been satisfied that the operative findings have vindicated my position and convinced them of the existence of this clinical entity.

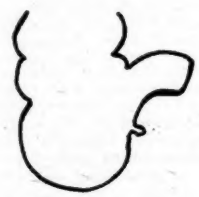

FIGURE V.
Tiny filling only.

FIGURE VI.
Not visualized.

The final proof lies in the relief from further attacks enjoyed by these children after the appendix has been removed, and many of these patients have been under observation now for from ten to fifteen years since operation.

In conclusion, while regarding this condition as such a definite entity, to be carefully sought for and confirmed before advising operation, I still thoroughly support the surgeon's condemnation of the indiscriminate removal of normal "lily white" appendices on the slightest pretext.

Finally, I maintain that the appendix which is the basis for the pain described as appendiceal colic is not a normal one, and, as I have just mentioned, the confirmation of this is evident in the relief of the symptoms after its removal.

Reviews.

Peripheral Circulation in Man: A Ciba Foundation Symposium. Editors for the Ciba Foundation, G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Jessie S. Freeman, M.B., B.S., D.P.H., assisted by Joan Etherington; 1954. London: J. and A. Churchill, Limited. 8″ × 5½″, pp. 232, with 72 illustrations. Price: 25s.

THE Ciba Foundation arranged a symposium, during May of 1953, on the peripheral circulation in man, and 32 physiologists, anatomists, physicians and surgeons were gathered from Europe and America to present results of their observations and to discuss each other's work. The results have been published in a Ciba Foundation monograph. Altogether 18 papers were presented, each with a discussion, and the field covered was wide. Detailed accounts are given of the most recent methods for measuring peripheral circulation in man, and some very ingenious apparatus is described. Blood flow in the skeletal muscles was considered as well as blood flow through the skin. U. S. von Euler and R. F. Whelan each give excellent accounts of the effect of adrenaline and noradrenaline on the blood flow through skeletal muscle and the differences between these two hormones. The response to cold and changes in temperature is discussed in several papers. The regulation of skin blood flow is considered by D. McK. Kerslake, the neurohistology by G. Weddel and W. Pallie, and the reflex control of cutaneous circulation by K. E. Cooper and D. McK. Kerslake. Other papers deal with vasomotor denervation, peripheral ischæmia in man, Raynaud's phenomenon, and functional disorders of the circulation. As would be expected from the calibre of the participants, the papers and discussions are of very high order.

While much of the contents of the book deal with physiology, there is quite a lot of discussion on the application of the findings to the study of disease processes in man, and any physician or surgeon who has to deal with disturbances of peripheral circulation in man will find much that interests him and much of value in diagnosis and treatment in this monograph.

Toxæmias of Pregnancy. By John Sophian, M.D. (London), F.R.C.S. (England), M.R.C.P. (London), M.R.C.O.G., with a foreword by F. J. Browne; 1953. London: Butterworth and Company (Publishers), Limited. Sydney: Butterworth and Company (Australia), Limited. 8½″ × 5½″, pp. 244, with 37 illustrations. Price: 35s. 6d.

THIS new work is the elaboration of the author's thesis that the toxæmias of pregnancy are precipitated by the Trueta mechanism which has resulted from increased uterine tension. F. J. Browne in the foreword remarks on the learning of the author, and as one reads the volume one is struck by the tremendous amount of reading, thought and research on animals which have formed the basis of the work. This is not green fruit hurriedly picked and packed to meet a favourable market; rather is it a basket of fruit picked by the grower at its prime and presented to a friend.

The volume deals with the anatomy, physiology, biochemistry, clinical signs, pathology and treatment of the toxæmias. One feels, however, that in his absorption in experimentation and theorizing the author has lost sight of the paramount practical importance of preventive treatment of severe toxæmia and eclampsia. For example, the chapter on treatment covers 23 pages, of which only half a page is given to prophylaxis, and in the summary (consisting of over two pages) of this chapter no mention is made of preventive measures at all.

The illustrations are generally clear and well placed in the text for easy reference. The general format, clarity of the print and freedom from errors reflect credit on the publishers. A comprehensive bibliography is appended.

The book is of course unsuitable for undergraduates, but will be bought by those studying for diplomas and by those obstetricians in practice who have the opportunity and the urge to delve deeply into the problems of their specialty. It should also find a place on the library shelves of hospitals and medical schools.

Synopsis of Pediatrics. By John Zahorsky, A.B., M.D., F.A.A.P., assisted by T. S. Zahorsky, B.S., M.D.; Sixth Edition; 1953. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 8″ × 5″, pp. 470, with 158 illustrations. Price: £3 18s. 9d.

IN presenting this "Synopsis of Pediatrics" the authors have not omitted any necessary detail; on the other hand, they have included some of the rarest diseases of children, the absence of which would not have been noticed by any

but the most critical reader. Therefore, the book will be of value to students during their term at children's hospitals, and then after graduation will be a most useful volume of reference for those who become general practitioners.

After introductory chapters dealing with such topics as the normal growth of infants, hygiene, nutrition and both natural and artificial feeding methods, the book goes on to itemize diseases of all the bodily systems. Congenital malformations are not forgotten, neither are the complex mental upsets of childhood, and brief mention is made of various skin, eye, bone and joint diseases. Diagnostic details for the various maladies described are clearly enumerated and treatment is laid down dogmatically, but at times perhaps too briefly. However, some clinicians will not agree with all the authors' therapeutics. Thus, in reference to poliomyelitis the Kenny method is advocated, whilst at the same time it is stated "we believe the child is best treated in the home"—surely an onerous task unless a staff of skilled nurses is employed.

Many practical procedures are illustrated by photographs, and in addition there are nine coloured plates illustrating the appearance of the mouth and throat in various infections, as well as showing examples of the appearance of normal and abnormal stools in infancy.

In this the sixth edition an appendix describing the treatment for acute medical emergencies is added for the first time, and the fact that over the past twenty years six editions have appeared proves the continued popularity of this very practical synopsis.

The British Contribution to Medicine. By Jaime Jaramillo-Arango, with a foreword by Arthur Macnalty, K.C.B.; M.D., F.R.C.P.; 1953. Edinburgh and London: E. and S. Livingstone, Limited. 9" x 6¼", pp. 232, with 45 plates. Price: 25s.

THERE can be no doubt that the British Commonwealth has a firm friend in the person of a distinguished South American, Dr. Jaime Jaramillo-Arango, former Rector of the National Faculty of Medicine of Bogota, whose literary work in the fields of medical science and history are characterized by thoroughness and erudition. While serving his country as Ambassador of the Republic of Colombia to Great Britain throughout the period of the Second World War, he went out of his way to make a survey of British achievements which have helped to promote the knowledge and progress of medical science. As a practical gesture in the cause of international understanding, and to make the facts available to the Latin-American people, he has embodied the results of his investigations in a book "The British Contribution to Medicine".

The opening chapter gives a general historical account of the more important scientific discoveries of Britishers who have helped to advance the medical, surgical, and allied branches of medicine since the beginning of the seventeenth century. The subsequent chapters deal exhaustively with modern research in the prevention of typhoid and paratyphoid fevers, malaria, and the nutritional diseases. In other chapters a full account is given of the steps which led up to the discovery and use of penicillin and the later antibiotics, and of the long-continued struggle to find the cause of cancer.

For its historical data alone this book should be of value to research workers in the various fields covered by Dr. Jaramillo-Arango's painstaking and strictly scientific investigation. The work is well documented throughout, although in many places the writing becomes too technical and abstruse to hold the attention of an average reader. There are a number of good portraits and illustrations, adding interest to the text; and Australians will note that their work has received more than a casual mention.

Experimental Diabetes and its Relation to the Clinical Disease: A Symposium Organized by The Council for International Organizations of Medical Sciences. Consulting editors, J. P. Hoet and F. G. Young; editors for the Council, J. F. Delafresnaye and G. Howard Smith; 1954. Oxford: Blackwell Scientific Publications. 9" x 6", pp. 362, with 54 illustrations, four in colour. Price: 35s.

MUCH work has been done during the past few years on the experimental production of diabetes by the administration of alloxan, anterior pituitary extract and other substances. Valuable material has thus been obtained for the study of the metabolic changes occurring in the disease. The relationship of the experimentally produced disease and the various kinds of naturally occurring diabetes is still a matter of study. A symposium on the subject was organized by the Council for International Organizations of Medical Sciences on the occasion of the first congress of the International Diabetes Federation, and twenty-two experts from Europe and America discussed various aspects of experimental diabetes. The results of their discussion have been published in a monograph entitled "Experimental Diabetes and its Relation to the Clinical Disease". Nineteen papers, each followed by a discussion, are given. The papers cover a very wide field. C. H. Best gives an excellent account of the islets of Langerhans with some fine coloured plates. Glucagon, the new pancreatic hormone, is discussed by C. de Duve and by J. L. R-Candela. There are three papers on alloxan diabetes by A. L. Lazarow, J. H. Gaarenstroom and P. Siderius, and by A. Loubatières. The relation of the adrenals to diabetes is considered by J. W. Conn, by F. Verzar, and by C. N. H. Long. Other subjects discussed are glycogen disease, growth hormone in relation to diabetes and to insulin, the action of insulin, diabetes and heredity, and pregnancy in relation to diabetes. R. D. Lawrence deals with the various types of human diabetes and their relation to experimental diabetes. Almost the best part of the book is contained in the discussions which serve to bring out points of difference in interpretations and difficult points not yet settled. Much of the recent work on the ætiology of diabetes has been brought together in this monograph and the whole is a stimulating production. It can be thoroughly recommended to any physician interested in the latest advances in the knowledge of diabetes.

Handbook of Differential Diagnosis. By Harold Thomas Hyman, M.D.; 1953. Philadelphia: J. B. Lippincott Company. Sydney: Angus and Robertson, Limited. 8" x 5¼", pp. 746. Price: 72s. 9d.

IT is usual to expect a "Handbook of Differential Diagnosis" to be a complete aid to diagnosis. However, the author of this compendium states in the preface that the work "does not claim to be inclusive or encyclopedic", and he goes on to justify his sacrificing of completeness for conciseness by stating that "a compendium in hand is worth many monographs in a remote library".

The opening pages are of blue paper to differentiate them from the rest of the text, and on these are listed alphabetically 1585 symptoms and signs, and each of these refers the reader to one or more headings or subheadings throughout the rest of the book.

The preface also states that "its style, chosen for utility rather than for literary merit, is not conducive to consecutive reading", a fact which becomes self-apparent after reading only a paragraph or two, for the brevity of many of the references make the book in parts read more like a dictionary than an aid to differential diagnosis.

The most comprehensive section is a chart summarizing a bulletin of the United States Department of Labour. This lists the various occupational hazards and their diagnostic features, and covers some 90 pages, and whilst undoubtedly this would be of great value to all industrial medical officers, to devote so much space out of 716 pages tends to make the book rather lop-sided, especially as many other important topics are incomplete. Thus, for example, under the heading of "Limp" no mention is made of the various internal derangements of the knee-joint as causes. Here and there the author interposes brief statements giving therapeutic advice, such as: "Prepare for urgent surgery", "Consider trial of aureo- or terramycin" and "Inaugurate vigorous antiinfective treatment". While these admonitions may be of admirable intent, yet in such a book as this they do seem superfluous.

Finally, under the term "Abulia" in the index the reader is referred to page 60 to find no mention there of this term which a medical dictionary defines as "a loss or defect of will-power", a state of mind induced soon after dipping into this volume, which will not easily displace from their place on medical bookshelves any of the better known works on differential diagnosis.

Resection-Reconstruction of the Hip: Arthroplasty with an Acrylic Prosthesis. By Jean Judet, Robert Judet, Jean Lagrange and Jean Dunoyer, edited by K. I. Nissen; 1954. Edinburgh and London: E. and S. Livingstone, Limited. 9¼" x 7", pp. 166, with 101 illustrations. Price: 30s.

THIS most informative little book serves a very useful purpose. It has been published at a time when controversy runs high amongst surgeons as to the merits and demerits of the comparatively new method of arthroplasty, in which the head or part of the head of the femur is replaced by an acrylic prosthesis to which is attached a stem consisting of acrylic with a core of stainless steel. This stem is hammered through a canal made in the remains of the head and neck of the femur, and its tip penetrates the lateral cortex of the femur.

The book gives a straightforward account of the use of this method in the treatment of osteoarthritis, certain fractures of the neck of the femur (including ununited fractures of this bone), traumatic arthritis, the sequelæ of septic arthritis, and congenital dislocation of the hip in adults. It describes results in a series of 400 arthroplasties performed by the Judet brothers and their team up to the date of publication of this book about two years ago. Since that time they have performed the operation on a further series of 450 patients.

The original series was commenced about eight years ago. There has thus been time to make a reasonable estimate of the early results of the operation. The results are assessed through a rather ingenious method of grading. The operation on patients suffering from osteoarthritis gave 17·1% excellent results, 47·8% of results were good, 19·2% were fair, and 15·7% bad. With regard to the durability of the prosthesis, fracture of the stem occurred in 3·5% of cases. All these accidents happened when true prostheses were not reinforced with a steel core.

The book is beautifully illustrated, with clear photographs and line drawings, and it includes a splendid set of photomicrographs of material removed at operation.

Physiological Foundations of Neurology and Psychiatry. By Ernst Gellhorn, M.D., Ph.D.; 1953. Minneapolis: The University of Minnesota Press. Geoffrey Cumberlege: Oxford University Press. Melbourne: Oxford University Press. 9″ × 6¼″, pp. 570, with 107 illustrations. Price: 89s. 3d.

In this book Professor Ernst Gellhorn, whose own researches on the nervous system have covered a wide field, has summarized recent laboratory investigations and discussed their bearing as clinical problems. Early in the first chapter he reminds his readers of the Adrian-Bronk law, "intensity of excitation is directly related to the frequency of discharge of the individual neurone and to the number of active neurones". In the restitution of movements after cortical lesions, reinforcement by acetylcholine and neostigmine, the increase of proprioceptive facilitation by eliciting reflexes such as the extensor thrust, and emotional excitement may all or severally be of value. In Chapter 7 there is a review of the effects of anoxia, hypoglycæmia, temperature and a variety of nociceptive impulses on the convulsive state. Sleep conduces to convulsions because of synchrony between slow large delta waves. *Petit mal* appears to be of subcortical origin since convulsive activity has been recorded from the diencephalon. For the understanding of consciousness attention in recent years has been focused on basal structures. The author suggests that normal cortical activity is a resultant of (*a*) exteroceptive and interoceptive impulses travelling along the long tracts; (*b*) activation of the posterior part of the hypothalamus by the facilitating system in the mid-brain; and (*c*) involvement of the dorsal medial nuclei of the thalamus by the diffuse afferent system of Morison and Dempsey in the reticular substance.

Part IV of the work is devoted to autonomic physiology, and here Dr. Gellhorn discusses the relationship between nervous and humoral activities, and reviews evidence that the rate of secretion of gonadotrophic, thyrotrophic and adrenotrophic hormones is regulated by the hypothalamus.

Psychiatrists will be specially interested in the pathology of the psychoses. The hypothalamus in schizophrenics usually gives inadequate responses in the sympathetic division, which may be boosted directly by electroplexy, or indirectly by insulin coma and barbiturate therapy through disinhibition of cortical control.

This informative work, based on a review of over 1200 references, including a hundred odd of Professor Gellhorn's own papers, will repay most careful study by research workers and clinicians in neurology and psychiatry if they bear in mind the limitations of applying the results of animal experimentation to human physiology, and especially to higher cerebral functions.

Emergency Surgery. Edited by Bernard J. Ficarra, A.B., Sc.B., M.D.; 1953. Philadelphia: F. A. Davis Company. Sydney: Angus and Robertson, Limited. 10½″ × 7″, pp. 1026, with 578 illustrations. Price: £9 13s. 6d.

This beautifully printed and illustrated book is written by some seventy specialists in different branches of surgery, radiology and anæsthesia. Consequently it embraces a wide field of emergency surgery, including some obstetric catastrophes. It has the disadvantage of being written by many authors, and different aspects of many of the subjects dealt with are thus discussed in different chapters so that continuity of thought is lost. For instance, the radiology of intestinal obstruction is dealt with in Chapter 23, its pathology in a part of Chapter 24, whilst its diagnosis and treatment are found in Chapter 30, each chapter having a different author.

One wonders at times on reading this work just what is implied by the term "emergency surgery". A long discussion of the ruptured intervertebral disk, including the technique of its removal, seems out of place in a book of this kind; and surely descriptions of several operations (not all satisfactory) for recurrent dislocation of the shoulder, or the reconstruction of ligaments in the acromio-clavicular or sterno-clavicular joints do not come within its province.

In a book such as this, written primarily for surgeons, it is not necessary to discuss such infections as rat-bite or cat-scratch fever.

Chapter 68, on bandaging, is redundant here and is better suited for a first-aid manual or a text-book for nurses.

Chapter 65, on the management of the injured following an atomic disaster, makes disappointing reading and seems to be a method of filling in twelve pages of the book for home consumption only. Chapter 66, on "Emergency Surgery for the Air Raid Casualty", is written rather for the pathologist or autopsy surgeon than for the emergency surgeon, whilst Chapter 67, on the radiation syndrome, is really of more interest for the physician. In fact, Section X (except for Chapters 63 and 64, on wounds and burns) could well be omitted.

This book cannot replace the classical succinct and less discursive books of other authors. The price of this work (£9 13s. 6d.) will limit its sale in the non-dollar countries.

Operative Surgery. By Guy W. Horsley, B.S., M.D., F.A.C.S., and Isaac A. Bigger, M.D., F.A.C.S.; Sixth Edition; 1953. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. In two volumes. 10½″ × 7″, pp. 1610, with 1274 illustrations. Price: £15 15s.

The sixth edition of "Operative Surgery" is certainly a worthy successor to the previous editions, the last of which appeared thirteen years ago. The paper, the printing and the illustrations are all excellent, and for those in need of a comprehensive text-book on operative surgery it is to be recommended. It is not, however, nor is it intended to be, a book for undergraduates. The width of the subjects covered is surprising and includes orthopædic surgery, genito-urinary surgery, neurosurgery, plastic surgery *et cetera*, and more details are given of these special branches of surgery than is usual in a general text-book on operative surgery. This applies especially to plastic surgery.

Since the previous edition appeared there has been a change in authorship of some of the chapters, and the intention expressed in the preface to the first edition of basing the book on the experience of the writers has been retained. References are given at the end of each chapter, but it is hoped that their selection, which is almost entirely from journals published in the United States, does not indicate a disregard for overseas publications. This bias in selecting references is especially important when statements such as "the British and others treat perforated ulcers by continuous gastric suction and antibiotics" are made and give a false impression of the popularity of such a method of treatment outside the United States. As is to be expected, numerous Americanisms appear among the English of the text without enhancing its clarity or beauty. For example, it is stated that "after the larynx has been skeletonized, the trachea is divided".

In a book of this size there must be many statements or omissions with which any single surgeon will disagree. Such controversial points include early ambulation in all cases after herniorrhaphy, the routine performance of bilateral oophorectomy in all women in the premenopausal period at the time of radical mastectomy for mammary carcinoma, a preference for œsophagocardioplasty rather than Heller's operation in most cases of cardiospasm, a bias against the upper mid-line vertical abdominal incision because it is subject to a greater transverse pull than is the paramedian incision, the failure to include splenectomy with total gastrectomy for carcinoma of the stomach, the failure to mention colectomy at the time of ileostomy in chronic ulcerative colitis, the failure to refer to the open methods of uretero-sigmoid anastomosis, the statement that kangaroo tendon sutures are preferable for repair of vesico-vaginal fistulæ, the inclusion of the horseshoe-shaped incision around the tip of the finger for the treatment of pulp space infections, the emphasis on the routine division and ligation of the superficial femoral veins in all non-fatal cases of pulmonary embolism, the omission of any reference to the radical vulvectomy of Stanley Way, the inclusion of a complementary caecostomy with resection and anastomosis of

the recto-sigmoid, the acceptance of the risk of pyæmic emboli to the liver in order to reduce the period of incapacity by immediate hæmorrhoidectomy in patients with prolapsed thrombosed internal hæmorrhoids, and the preference for suprapubic rather than retropubic prostatectomy. Although the disadvantages of the perineal method of prostatectomy are given, it is of note that its technical details are described in full with nine illustrations, whereas only two inadequate illustrations of the retropubic method are included.

Despite these minor criticisms, this would appear to be one of the best standard works on operative surgery, and it can be thoroughly recommended to all who require such a book for systematic post-graduate study or as a reference book when details of an unfamiliar operation are required.

Sex in History. By G. Rattray Taylor; 1953. London: Thames and Hudson. 8¼" × 6", pp. 344. Price: 21s.

BY calling his book "Sex in History", G. Rattray Taylor appears to have bitten off more than he can chew, though he admits that it is more like a primer than a full study of the subject which might occupy many volumes. Unlike many other works dealing with the same subject as it may have existed in other countries and in other civilizations, this is not a descriptive work. It is essentially critical, and within the compass of three hundred pages it sets out to trace the effect of religion—and more particularly the Christian religion—upon the sexual life of man. Rightly he accuses most historians of neglecting this subject; and rightly again, he claims that it cannot be overlooked if man is to be seen in his true perspective. For our sexual code, he says, is a "strange and arbitrary amalgam" of magic, make-believe, witchcraft and religion. Our ideal is not built upon the unequivocal teaching of Christ, but upon the neurotic guilt feelings of a collection of misguided and repressed "church fathers" who did not scruple to place their own interpretation upon Christ's words, but often went further and introduced their own dogmas under the guise of divine teaching.

The burden of this book rests under the belief that social ideas are conditioned by parental introjection. Where the community is largely modelled on the mother figure—a matrist community—there is a more permissive attitude to sex, the status of women is elevated, hedonism and pleasure are welcomed. But when father identification has taken place—a patrist community—there is a restrictive attitude to sex, an authoritarian political set-up, chastity is worshipped, women are subjected, and asceticism and fear of pleasure predominate. By and large, sex in history, therefore, reacts in relation to the swing of matrist and patrist cultures. There is more to it than this, of course; there are cultures in which sex has been a sacrament, and in which it has existed as a shame and finally as a guilt. But the author's somewhat melancholy conclusion is that we are born amid the wreckage of an old myth, and "seem unable to escape from the tyranny of our obsessive demands, to serve which we have created a mode of life wherein the direct satisfaction of instinctual needs has become increasingly difficult".

This book should be full of interest to the sociologist and should be a stimulus to anyone who is interested in history and in human behaviour.

Headache: Diagnosis and Treatment. By Robert E. Ryan, B.S., M.D., M.S. (in Otolaryngology), F.A.C.S.; 1954. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 9" × 6", pp. 338, with three illustrations. Price: £3 8s. 3d.

IN this book Dr. Ryan has endeavoured to differentiate types of headache. He gives his views on the vascular type of headache. In his view, dilatation and constriction of the cranial blood vessels are the main cause. He describes histamine headache as being of sudden onset, great severity and sudden offset. He arrives at the diagnosis by injecting histamine intravenously. For treatment he recommends a daily injection of histamine until the headache ceases. For migraine (which he describes in detail), he recommends also histamine injections. For the immediate relief of most headaches he recommends ergotamine tartrate, with or without caffeine. "Cafergot" is a preparation containing the above. Tension headache is his name for what might be called nervous headache. The author describes also psychogenic headache, sinus headache, myalgia of the head and mixed type of headache. He deals with other head pains due to ear diseases, meningitis, neuralgia, cerebral tumour and other organic diseases. Naturally, allergic headaches are alluded to. These appear to be rather difficult to differentiate. In general, Ryan has given quite a useful résumé of the causes of headache. So far as treatment is concerned, apart from the tedious daily injections of histamine, there is little new.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Transference: Its Meaning and Function in Psychoanalytic Therapy", by Benjamin Wolstein, Ph.D., with an introduction by Clara Thompson, M.D.; 1954. New York: Grune and Stratton. 8" × 5½", pp. 220. Price: $5.00.

The author has tried "to set forth a new perspective in which to consider the analysis of transference as a therapeutic instrument".

"A Synthesis of Human Behavior: An Integration of Thought Processes and Ego Growth", by Joseph C. Solomon; 1954. New York: Grune and Stratton. 8½" × 6", pp. 278, with three text figures. Price: $5.50.

The author "has attempted to categorize human behaviour according to the growth of ego development".

"Advances in Enzymology: And Related Subjects of Biochemistry", edited by F. F. Nord; 1954. New York: Interscience Publishers, Incorporated. London: Interscience Publishers, Limited. Volume XV. 9½" × 6¼", pp. 558, with 56 text figures. Price: $11.00.

There are twelve authors of this book and twelve sections, one of which is in French and one in German.

"CIBA Foundation Symposium on the Kidney: Arranged Jointly with the Renal Association." Editor for the Renal Association, A. A. G. Lewis, B.Sc., M.D., M.R.C.P.; editor for the CIBA Foundation, G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., assisted by Joan Etherington; 1954. London: J. and A. Churchill, Limited. 8" × 5½", pp. 350, with 125 illustrations. Price: 32s.

A verbatim account of the symposium held at London from July 7 to 10, 1953.

"Medicine for Nurses", by W. Gordon Sears, M.D. (London), M.R.C.P. (London); Sixth Edition; 1954. London: Edward Arnold (Publishers), Limited. 7¼" × 5", pp. 528, with 80 illustrations. Price: 16s.

The first edition was published in 1935.

"Spinal Epidural Analgesia", by P. R. Bromage, M.B., B.S. (London), F.F.A.R.C.S., D.A.; 1954. Edinburgh and London: E. and S. Livingstone, Limited. 9" × 6", pp. 130, with 41 illustrations. Price: 15s.

Based on the records of 1000 "personal administrations" over a period of five years.

"Living Happily with a 'Heart'", by Tom Clarke; 1954. London: Victor Gollancz, Limited. 7½" × 4½", pp. 132, with one illustration. Price: 7s. 6d.

The author is a journalist and writes of his own experiences.

"Study Guide for Clinical Nursing: A Co-ordinated Survey Integrated with Essentials of the Basic Sciences", prepared under the direction of Emily C. Cardew, R.N., M.S.; 1953. Philadelphia, London and Montreal: J. B. Lippincott Company. 9" × 6", pp. 576. Price: 64s. 6d.

The book is described by the editor as "a student's working tool designed to aid in acquiring the knowledge and the skill basic to the provision of complete nursing care of the patient".

"Cardiovascular Surgery", by Gerald H. Pratt, M.D., F.A.C.S.; 1954. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9½" × 6", pp. 844, with 358 illustrations. Price: £8 1s. 3d.

The author's purpose is "to bring to surgeons, internists and students a summary of acceptable treatment for cardiovascular lesions".

"Geriatric Medicine: Medical Care of Later Maturity", edited by Edward J. Stieglitz, M.S., M.D., F.A.C.P.; Third Edition: 1954. Philadelphia: J. B. Lippincott Company. Sydney: Angus and Robertson, Limited. 10" × 7¼", pp. 742, with 205 illustrations. Price: £8 1s. 3d.

The book is divided into eight sections and comprises 42 chapters. There are 48 contributors.

The Medical Journal of Australia

SATURDAY, AUGUST 28, 1954.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE CHILD IS THE FATHER OF THE MAN.

IT was Wordsworth who wrote that the child is the father of the man. Milton had the same idea in "Paradise Regained" when he declared that

> The childhood shows the man,
> As morning shows the day.

This idea should be in the mind of every thoughtful citizen of today when he thinks of the accelerated pace of modern life with its hurdles and its hazards, with its calls on man's physical endurance and with its demands on his moral rectitude and his constancy. The present issue of the journal is devoted to the papers and discussions which took place at the annual meeting of the Australian Pædiatric Association. These articles are not to be regarded merely as another series of medical papers for the consumption of the practitioner. They should be looked on as dealing with the promotion of healthy childhood so that it may be followed by robust bodily and mental development in adult life. No department of medical practice should be considered as a single entity standing alone. If we are to be logical, we must think of the conditions under which children are conceived, of the health of their mothers during pregnancy, of the actual birth of the child and its care in infancy, of its health as it grows and of its mental development while it is growing, of its training to become a useful citizen; and then we must turn our attention to the young man and the young woman, regarding them first from the point of view of preventive medicine, and when that fails, from the standpoint of curative medicine. In other words, we must realize that pædiatrics takes its place between obstetrics and adult medicine. We know from everyday medicine that a diseased and degenerate father cannot, in the ordinary course of events, have a healthy and happy, let alone a useful, child. We also know that a healthy and happy child is likely to become a healthy and useful man. And here we may call to mind what Wordsworth and Milton wrote. It may be useful, therefore, to consider what medicine can do to promote the general health and welfare of children.

To point out that the physical care of children is preventive and curative is to state what is obvious. On the preventive side, measures may be adopted by the practitioner or by the State. Preventive pædiatrics is not the sole province of the specialist pædiatrician. Far from it. The family doctor is the one who has more opportunity than any other practitioner to direct a child's parents in the steps of disease prevention. It is clear that prevention of disease in children must start with the pregnancy of the mother. This has become widely recognized ever since the work on rubella occurring during the pregnancy of mothers and on the possibility that their offspring may suffer from various congenital defects. In the early life of the child the practitioner is often consulted about the matter of feeding, and he is, or should be, well able to take charge in such matters. In the prevention of infectious disease he often has to advise the parents about prophylactic injection, and he has to be prepared to give directions about the child's attendance at school or on other collective occasions. The discussion at the Pædiatric Association's meeting on the problem of the deaf child will help to bring home to the practitioner the importance of discovering deafness at an early stage in the child's life. If he is in any doubt in the matter he should always seek the help of somebody experienced in the diagnosis of deafness in children. The supervision of a child's health must not be confined to the earliest years of his life. It should extend into his adolescence. The State takes a large part in this matter, as indeed it should. The Commonwealth Government some years ago established what are known as the Lady Gowrie Child Centres. This was done with the cooperation of the Federal Organization of the Kindergarten Unions, the whole organization operating under the title "The Australian Association for Pre-School Child Development". One of these centres is in existence in each of the capital cities. Recently, the Lady Gowrie Child Centre committees have been given a greater degree of autonomy, and the management of each centre is now in the hands of a local committee. In 1950-1951, the Commonwealth Government spent on child centres the sum of £25,000. In the States, baby health centres or welfare centres exist, and babies may be taken to these centres where advice and guidance are given to mothers free of charge. When the child is older and goes to school, he comes under the influence of the health inspection system of the State Education Department. In these departments work of the utmost use is done, but many of them are understaffed and it is recognized that children should be examined for abnormalities oftener than they are. A health measure which should be mentioned is that by which holidays are provided for children at the seaside or at health resorts. In some States, camps for school children are arranged, and in New South Wales the Far West Children's Health Scheme brings to the seaside in the summer children who have never seen the sea and have never known the joy of playing on a sea beach. In addition to, or perhaps in spite of all these government undertakings, emphasis must be laid on the over-all

importance of the work of the family doctor. His activities in the early years of a child's life have been mentioned and he should exercise his supervision until the child has passed out of childhood into the adolescent stage. Whether the practitioner is able to do this will depend largely on the outlook of the parents, but even so, the practitioner will have ample opportunity to instruct parents on what should be done.

On a child's mental health a great deal might be written. Nowadays the newspapers are full of reports of juvenile crime and wrongdoing of various kinds; the recent reports of murder in New Zealand by two girls fifteen years of age have horrified the whole Commonwealth. Theft and vandalism by children are all too common. Much of the blame for these things must be laid at the door of the parents or guardians, some of whom no doubt set for their children an example in wrongdoing. Pope wrote that "just as a twig is bent, the tree's inclin'd", and we also read in the book of "Proverbs" that if we train a child in the way he should go, when he is old he will not depart from it. The medical practitioner has not the duty of bending the twig, of training the child in the right way—unless the child is his own. He may be the medical officer in charge of an institution that deals with recalcitrant children, in which case he can join with others in an attempt to unbend the twig. The pædiatric specialist probably has more opportunity of activity in the direction of difficult children. What the family doctor and the specialist can do is to encourage or even insist on the adoption of healthy habits of play and exercise by children of all ages. There is no doubt that games played in the right way—they have to be taught if this is to be done—will have an enormous effect on the future outlook and behaviour of the child. This naturally introduces the question of where the children are to play. In some Australian cities, children have nowhere to play but the streets. It is small wonder that in some instances bad habits are created and evil associations are formed. In all this discussion, we have to bear in mind that the child of today is older for his years than he was, say, fifty years ago. This is in large measure the result of pleasures and distractions which are provided for him—the cinema, the comic strips in the newspapers, serials on the radio, the treats arranged for him, and the lax supervision by parents who do not know, who do not care, or who cannot be bothered. The subject of parental control is outside the scope of the present discussion; unfortunately it is at the root of the present unrest and turbulence among the childhood of today. The problem is one which concerns the whole community, and we have to remember that we have it on the highest authority that it were better for a man who offends one of the little ones that a millstone were hanged about his neck and that he be cast into the sea. The final conclusion of the present discussion is that pædiatrics lies at the root of medical practice. It may well be regarded as the specialty of specialties, for on the success of its practice and the adoption of its principles lies the whole future of our nation and race.

Current Comment.

SURGERY OF THE NEWBORN.

So many articles on the medical side of pædiatrics are published that some practitioners may perhaps forget that surgery is a branch of pædiatrics as well as medicine. It must, of course, be remembered that advances in pædiatric medicine may be applied to surgery as practised in the treatment of children and infants. The reverse holds true, and we have only to think of the advances in cardiac surgery to realize the truth of this statement. The way in which surgery of the newborn has progressed is clearly shown by a recent report on 71 cases occurring in the years 1942 and 1952. The report is by W. H. Snyder, junior, M. H. Snyder and L. Chaffin, of Los Angeles.[1] The report by these authors was read at the annual meeting of the Western Surgical Association held at Chicago in December, 1953. At the Children's Hospital in Los Angeles there has been an infants' ward since 1927. The authors of this report took two sample years, a decade apart, and considered the infants operated on at this institution. The years chosen were 1942 and 1952. Fifteen newborn babies were operated on in the year 1942; these were under two weeks of age. The babies operated on in 1952 numbered 56, making a total of 71. It was found that the percentage of newborn babies in the hospital population had not increased over the ten-year period. In 1942, 1317 infants under two years of age were admitted to the Los Angeles Children's Hospital, and 174 of these, or 13%, were newborn babies under two weeks of age. In 1952, 2496 infants were admitted, and 304, or 12% of them, were newborn. However, the percentage of these infants receiving treatment doubled, and the mortality following surgical treatment was almost halved. It is pointed out that when a newborn baby is seen by a surgeon, he is a few hours or a few days old. He is unlike older infants in many respects. Readjustments in breathing, heat regulation, circulation and fluid balance have to be accomplished. Most of these changes are made with in a few hours if the baby is a full-term baby, but they require many days or weeks if he is premature. Of the series of 71, 30% were premature or weighed under five and a half pounds. The average weight was only six pounds. Special points in management are discussed. First of all, the newborn baby requiring surgical operation must receive aid in the stabilization of his heat-regulating mechanism by means of proper clothing or by incubator control. Of the group of infants operated on in 1952, more than half were placed in incubators; none of the 1942 group was so treated. In the second place, an atmosphere with a high oxygen percentage will aid the respiratory and circulatory systems in effecting an adjustment to extrauterine life. The need for oxygen is almost directly proportional to the degree of immaturity. Only one-third of the 1942 group received oxygen, but nearly three-quarters of those in the 1952 group benefited by it. Moisture has only recently been added, but it is now common practice to use it in the form of "Mistagen" with the administration of oxygen. In the third place, fluid requirement of the newborn calls for special clarification. No fluid whatever is essential during the first two days of life. The organism is so constituted that this period of adjustment is naturally provided for. We learn that water constitutes 90% of the six-week embryo, 91% of the five-month fœtus, and 80% of the newborn contrasted with 72% of the adult.

In 1942 newborn infants treated surgically were often given fluids in amounts comparable with the requirements of older infants. On the basis of 75 millilitres per pound, this meant that 450 millilitres were given to a six-pound baby in twenty-four hours. It is now realized that this is at least twice the amount required, and that, furthermore, fluid administration should not be started until the third day. At present it is believed that the total amount of fluid given parenterally should be nil in the first three

[1] *Arch. Surg.*, April, 1954.

days except for replacement of actual measured loss from vomiting or blood loss. After this, 30 millilitres per pound of dextrose in water is ample for a twenty-four hour period. The amount of saline solution used has been rigidly curtailed in 1952, whereas in 1942 it was the main fluid used parenterally. In premature infants no intravenous saline therapy is given, and for the more mature infants less than one-fourth of the total amount of fluid contains salt. In the 1952 period antibiotics and vitamins, including vitamin K, have been given as a routine measure to newborn infants. The anæsthetic agents that have been used have changed. In the earlier period, 100% of the administrations consisted of ether; in 1952, the figures were 57% ether and 43% cyclopropane. In the later period, a combination of several anæsthetic agents was used in several cases. It is of interest to note the surgical conditions for which operation was carried out in the two periods. Thus in 1952 there were twelve cases of œsophageal atresia with fistula, nine of intestinal atresia, six of deformity of the lip and jaw, five of imperforate anus, five of malrotation, five of omphalocele, four of obstruction to various parts of the alimentary tract, four of malignant disease, three of trauma to the larynx and intestine, one of diaphragmatic hernia, one of meningocele and one of perforation of the bladder. In 1942 there were two cases of œsophageal atresia, seven cases of intestinal atresia, one case of omphalocele, three cases of intestinal obstruction, one case of malignant disease and one of meningocele.

Snyder and his co-workers describe the types of operation which were performed, and give details of the way in which some of them were carried out. These need not concern us at the moment. It will suffice to show that the progress made in surgery of the newborn has been considerable, and it is probable that the success attained by these authors in Los Angeles is reflected in other surgical centres.

THE AFTER-EFFECTS OF ANÆSTHESIA ON CHILDREN.

ANYONE who has had much experience with the treatment of children will realize the enormous variation shown by them after they have undergone different therapeutic procedures. Some children take kindly to hospital and others do not. The type of reaction manifested by them is partly the result of the consideration and treatment which they receive in hospital, and arises partly from the way they have been treated at home and have been prepared for the hospital ordeal. Nowhere is this more manifest than in connexion with the induction of anæsthesia for various operative procedures. There comes to mind a sturdy little boy who had to face an operation and was anæsthetized by a senior resident medical officer at one of the teaching hospitals. This took place some years ago. The anæsthetist could not pronounce his "r's" or "l's" properly, but turned them all into "w's". After the child had recovered from anæsthesia, the ward sister asked him how he got on. "Oh", said the child, "all right. He said, 'bweave in' and I bweaved in, and he said 'bwow out' and I bwowed out." Unfortunately, all children are not like this little boy. Several articles have been written in late years on the effects of anæsthesia in children. In 1945 David M. Levy published an interesting study on the psychic trauma of operations in children.[1] Levy thought that operations should be postponed if possible till the child was at least three years of age, especially if evidence of anxiety or of undue dependence on the mother was present. He said that an explanation should be made to the child of what was about to take place. Contact with the mother, in his opinion, should be made both immediately before and after the operation. He held that a preparatory sedative and anæsthetic should be given in the bedroom in order to spare the child the experiences of seeing instruments, the operating theatre, and so on. J. E. Eckenhoff has written on the pre-anæsthetic sedation of children before tonsillectomy and adenoidectomy.[1] He discussed the various drugs which should be used before anæsthesia was induced. He thought that the incidence of crying before anæsthesia could be minimized by the addition of a barbiturate to a dose of belladonna. He said that crying increased the hazard of anæsthesia. He was opposed to the use of morphine. Eckenhoff has now published another article, entitled "Relationship of Anesthesia to Post-operative Changes in Children".[2] He states that no attempt has previously been made to relate the experiences of the induction period of anæsthesia to the development of undesirable personality changes afterwards. He points out that it is difficult to establish precise relationships because the experiences of admission to hospital, of anæsthetization and of operation are not easily separated. Over a period of eighteen months he sent to the parents of all children admitted to the Hospital of the University of Pennsylvania for an otological operation a *questionnaire*. The *questionnaire* had to do with enuresis, night cries or terrors, temper tantrums, fear of meeting strangers, fear of having the face covered, fear of new odours. He asked that the replies to these questions should be related to the child's experiences in hospital. He also asked how the child had been prepared for admission to hospital and operation. Letters were sent to 1008 parents and replies were received from 612, or 61% of the total. Parents of children twelve years of age or older were less interested in the study than others. When this group was deleted from the study, the replies to the *questionnaire* were 65% of the total. In his study, pre-anæsthetic medication consisted of the administration of pentobarbital and scopolamine and usually morphine in doses recommended by M. D. Leigh and M. K. Belton, who have written on this subject. Anæsthesia was administered by a resident medical officer or a staff anæsthetist, by a medical student or by an intern under supervision. Of the replies, 17% indicated that a personality alteration had occurred which might be traced to anæsthesia or to the hospital experience or to both. Of the changes, 32% were in the form of night cries or terrors, appearing for the first time or becoming worse after the child returned from hospital. Temper tantrums comprised 26% of the changes, fear phenomena constituted 23%, and the remainder (19%) had to do with bed-wetting. Eckenhoff found, as Levy also found, that incidence of personality change was highest in the young children. He sets his results out in a table and states that they are as clear-cut as those of Levy. The percentage of personality changes was approximately the same with all types of induction agents. One surprising finding was that of 25 complaints related to bed-wetting, 23 occurred in patients who had been given vinyl ether and only two after the use of other agents. On a percentage basis, bed-wetting was five times more common after the administration of vinyl ether as an induction agent. The finding is regarded as the more interesting since there was no remarkable difference in other changes compared with the anæsthetic agent administered. Some short case histories are quoted. In one instance the mother complained that the sedative was not given to the child soon enough before he was taken to the operating room, and he was terrified. Another reported that the child had nothing but a pleasant recollection of the operation. The mother attributed this entirely to the drug that had been given to him before he went to the operating room. Another child filled in her time after operation by playing with other children at operating theatre procedure. She was always pretending that she was giving her dolls ether, and the mother had to hide her needles because the child took them and stuck them into persons who were in the house. This child underwent several operations. In the first instance her premedication was entirely satisfactory. In the second instance it was not, and the child became terrified. Afterwards she suffered from screaming tantrums. The mother declared that before the child had another operation she was going to have a clear understanding with the doctor that medication was to be fully carried out beforehand. Two instances are given in which the child was told before

[1] *Am. J. Dis. Child.*, 1945, Vol. 69, page 7.

[1] *Arch. Otolaryng.*, April, 1953, page 411.

[2] *Am. J. Dis. Child.*, November, 1953, page 587.

she went to hospital stories which were not true. Eckenhoff admits that one cannot blame all personality defects or abnormalities on unfortunate experiences in hospital. At the same time, he insists that his study and those of others suggest that the experiences may be important. With this most clinicians will agree. In Eckenhoff's previous study he concluded that there was a clear-cut relationship between the type of pre-anæsthetic medication and the incidence of crying before the induction of anæsthesia. He had reasoned that the crying child might be the one in whom personality changes were likely to develop, and he concludes from the present study that this reasoning was correct. He can find no explanation for the association of bed-wetting with the use of vinyl ether. His general conclusion that unsatisfactory pre-anæsthetic medication and induction of anæsthesia are associated with a high incidence of personality change in young children must be accepted. Eckenhoff's study is concerned chiefly with pre-anæsthetic agents. It is to be noted that he does not lay much stress on the experience of the anæsthetist. This surely is of the utmost importance, and it is reasonable to insist that full and proper supervision should be exercised when inexperienced anæsthetists have to administer anæsthetic agents to children.

PINK DISEASE AND GANGLION-BLOCKING AGENTS.

MANY have felt that in pink disease there is a profound disturbance of the autonomic nervous system. It is strange that so little experimental effort has been directed toward this aspect of the disease. B. D. Bower,[1] at the Birmingham Children's Hospital, has recently investigated this matter. When part of the body of a normal adult is heated so that warmed blood, and perhaps afferent nerve impulses, reach the hypothalamus, the result is vasodilatation. Arteriolar tone is inhibited and possibly vasodilator fibres are stimulated. The classical way of measuring this vasodilatation is by measurement of skin temperature. This response has been demonstrated many times in adults, but little work has been done on children. Bower therefore set about establishing a normal standard in children. The method used was to make the child lie on a canvas sheet through which one arm projected and was immersed in water kept at a temperature between 42° and 44° C. Skin temperature was measured by thermocouples attached to the skin of the other limbs. In fifteen normal children ranging in age from three months to thirteen years the response resembled that in the adult, a prompt rise from about 22° to about 36° in ten to twenty minutes. In twelve infants in the first two weeks of life the response was much more sluggish, the skin temperature rising from about 24° to about 32° in sixty to seventy minutes. Between the ages of two weeks and three months there was a gradual change from the infantile to the adult response.

Bower then investigated the reaction of thirteen children suffering from pink disease. In all the response was quite abnormal. In seven there was no rise at all in skin temperature, in four there was a late small rise in one limb and in two a small rise in all three limbs. All children sweated profusely during the experiment. He repeated the test on each child after an intramuscular injection of hexamethonium bromide or pentapyrrolidinium bitartrate ("Ansolysen"). Strikingly different results were obtained. Vasodilatation could now occur, for the temperature rose, fairly normally in the lower limbs and rather less so in the arms. But in addition to this other changes were noted: sweating was markedly reduced; the hypertension so characteristic of pink disease was lowered within twenty to forty-five minutes of the injection; and the children, who formerly had been whimpering, miserable and restless, lay quiet and good, or even slept.

This work confirmed the autonomic disturbance of pink disease, but suggested too that the autonomic ganglion-blocking agents might be useful for therapy. Bower went on and treated these children for several weeks with one or other of these drugs. The initial dose of hexamethonium bromide given was five or ten milligrammes by hypodermic injection four times daily, the dose being increased gradually if necessary up to 15 to 20 milligrammes. Of "Ansolysen" the initial dose was one milligramme by hypodermic injection four times daily with increments of 0·5 milligramme up to as high as three milligrammes. He gives clinical reports of the children treated, and there seems little doubt that great benefit resulted. The main effects were relief of sweating, of hypertension and of the vasomotor changes in the hands and feet, and the very great improvement in the child's demeanour. One mother described the change in her child as a miracle. When treatment was stopped after a week relapse occurred, but after three weeks of treatment improvement was maintained when the drugs were withheld. Bower has recently been using "Ansolysen", which he regards as the better drug, by mouth,[1] commencing with a dose of five milligrammes four times daily, and making increases of five milligrammes as necessary. The best dose for each child has to be determined by trial, as is the case when these drugs are used for the relief of hypertension in adults. Bower is modest in his claims for this new therapy. He has, however, demonstrated that a gross disorder of the autonomic nervous system does exist in pink disease and that this disorder can be influenced by drugs. One might expect sweating, hypertension and peripheral vasomotor changes to be affected. The relief of the intense misery of these infants probably follows the establishment of a normal circulation in their hands and feet, for the occasional child with pink disease who is old enough to describe his symptoms will complain of very real discomfort in these parts—a burning, itching sensation.

Many questions remain to be answered. First, perhaps, is this therapy free of danger? Bower apparently found none. It is known that some adults are more sensitive than others to these drugs, and that fall in blood pressure and collapse may follow ordinary initial doses. Could the same apply in infants, and could such collapse be dangerous in a sick hypotonic infant suffering from a disease in which death from sudden circulatory failure is by no means rare? We still must find, too, the most suitable initial dose and the best interval between doses. And what criteria of optimum dosage should be used? In two children treated since Bower's work has become known, it seemed that lessening in sweating and vasomotor changes and improvement in demeanour occurred with doses that reduced the blood pressure, but not nearly to the normal level. And how long should treatment be continued? If, as Bower's case notes suggest, three weeks is often long enough, then perhaps this treatment is more than just symptomatic relief, for one would have expected that without treatment the illness would persist long beyond this period.

Whatever the ultimate assessment of Bower's work, it is of the utmost interest, and certainly deserves extensive trial.

[1] *Quart. J. Med.*, April, 1954.

IDIOPATHIC HYPERCALCÆMIC SYNDROMES OF INFANCY.

THE systematic biochemical examination of blood and other body fluids has led to the discovery of several new diseases. Two of these are associated with a large increase in the calcium content of the blood in infants. In 1952 Fanconi and Girardet, of Zürich, with Schlesinger, Butler and Black, of London, described two young children with a hitherto unrecognized syndrome of physical and mental retardation, chronic renal disease, osteosclerosis, and persistent hypercalcæmia. Schlesinger, Butler and Black concluded that "the disorder is due to a collection of separate defects, probably genetically determined, resulting in congenital abnormalities affecting heart, kidney, skeleton

[1] Personal communication.

and metabolism". Several cases have been described since. Again in 1952, Lightwood and also Payne described a similar but not identical conditions under the title of "idiopathic hypercalcæmia of infants with failure to thrive".

K. G. Lowe, J. L. Henderson, W. W. Park and D. A. McGreal have described in considerable detail one case of the first condition and briefly six cases of the second condition. R. D. G. Creery and D. W. Neill have described sixteen cases of "idiopathic calcæmia of infants with failure to thrive".[1] Calling the first condition Syndrome II and the second condition Syndrome I, Lowe *et alii* note the principal features in which the two conditions differ from or resemble each other. Syndrome I is transient and benign. Recovery begins after a few weeks and there is no permanent physical or mental impairment. Syndrome II is prolonged and severe, with much retardation of physical and muscular growth. Even with improvement in the general condition the mental state usually remains poor. The facies is normal in Syndrome I, but in Syndrome II it may be characteristic with wide mouth, thick upper lip and receding mandible. In Syndrome I there is no hypertension and no cardiac bruit, while in Syndrome II hypertension and loud systolic bruit occur. The bones are normal in Syndrome I, but radiography shows osteosclerosis in Syndrome II. The serum calcium content is high in both conditions, 12 to 18 milligrammes per 100 millilitres, as also is blood urea content. The case of Syndrome II was studied by Lowe *et alii* in considerable detail over a period of nearly two years, when the child died. No form of treatment tried had any beneficial effect. The six patients with Syndrome II studied by Lowe *et alii* showed symptoms within the first six months of life, and all but one, who died of bronchopneumonia following chicken-pox, were well on the way to recovery after a few months' treatment. In Creery and Neill's sixteen cases the onset of definite symptoms was usually clearcut at ages from six weeks to nine months. In one infant symptoms were present from birth. All the infants failed to gain weight normally, and some were four to five pounds below their expected weights. Physical examination showed little apart from wasting. Most of the infants were alert and contented. The pleasant nature, associated with a diminutive appearance, sunken eyes and a slender neck, has sometimes suggested the diagnosis. All showed a blood calcium content of over 12 milligrammes per 100 millilitres, up to 17 milligrammes. Inorganic phosphate figures were within the normal range for children of this age. Radiographs of the wrist were taken in 15 cases; these were normal in three cases, but 12 showed transverse bands at the metaphyseal margins of the lower ends of the radius and ulna. These bands were single, narrow and well defined, similar to the lines seen in lead poisoning. Most of the children recovered without any treatment other than the cutting down of their vitamin *D* intake. In several cases a diet low in calcium content was given; the results were inconclusive except in one case which showed a gratifying response. In none of the cases of either series had excessive doses of vitamin *D* been given, but in most more than usual had been given, and there was some evidence that this caused a greater absorption of calcium from the intestine than usual. Most of the symptoms are probably related to the hypercalcæmia *per se*.

The only constant ætiological factor is a high calcium intake from early infancy, usually associated with a more than adequate amount of vitamin *D* in the diet. Lowe *et alii* consider that excessive administration of alkaline medicines, such as milk of magnesia, may have been a factor in some but not all the cases. No ætiology can, as yet, be given for the more severe Syndrome II. It may be a severe form of Syndrome I, but this is unlikely. The milder condition must be quite a common disorder. Forty-two cases have been reported in Great Britain in the past year, and many more have been recognized. As the editor of *The Lancet* asks in a leading article: "Is this the price we pay for the abolition of rickets?" The editor also suggests that the disease may have been with us masquerading under such names as marasmus, feeding difficulty and "teething"; and the tendency to spontaneous recovery would lead to credit being given to various forms of treatment, and especially to changes in feeding.

[1] *Lancet*, July 17, 1954.

CLOSURE OF THE DUCTUS ARTERIOSUS IN THE INFANT.

THE change-over from fœtal to post-fœtal circulation presents problems of exceptional interest. One of these is the closure of the *ductus arteriosus*. Barclay and his co-workers, also Barcroft, were of the opinion that closure in lambs occurred a few minutes after birth; later investigations with a radioactive "tracer" threw doubt on this conclusion and indicated that the cessation of flow from the pulmonary artery to the aorta took place gradually and was not completed until several days had passed. Certainly the heart of the calf obtained from the slaughterhouse displays an obviously patent ductus, but the calf when born does not present any signs of circulatory defect; one surmises in this case that muscular spasm obliterates the lumen at birth. A recent study by F. L. Eldridge, H. N. Hultgren and M. E. Wigmore[1] was stimulated by the observation that numerous infants show definite cyanosis of the lower part of the body but not of the upper portion. Such difference is well known in children and also in adults when patency of the ductus is definitely proved to exist. In this investigation 12 newborn infants were subjected to an examination of the oxygen content of the blood obtained by special technique from the right hand and a foot. In all cases a marked difference was obtained, greater than could be accounted for by experimental error. The right hand blood was arterial; the foot blood showed venous admixture. After three days no difference could be detected in most of the infants, a fact pointing to complete closure of the *ductus arteriosus*. In one case identity of the specimens of blood from the two sources was apparent only on the fifth day. Further studies on this subject are being carried out in Stanford University School of Medicine with a view to determining more accurately the periods of partial and complete closure and what factors influence these happenings.

TREATMENT OF LEAD POISONING.

EDATHAMIL CALCIUM-DISODIUM is a recent and apparently promising addition to the hitherto disappointing medicaments suggested for the treatment of lead poisoning in children. According to R. K. Byers and C. Maloof,[2] it is soluble in water and in the usual watery solutions used for intravenous injection. It forms un-ionized soluble very stable complexes with metallic ions. Given simultaneously with lead, it protects an experimental animal from large doses of lead, though the dosage of lead can be enlarged beyond the effective limit of the edathamil. Given to animals already given lead, it removes most of the lead from the soft tissues, but not a great deal from the skeleton. Excretion then apparently takes place through the urine. Clinically edathamil is given intravenously in dextrose solution. Byers and Maloof give details of the dosage scheme as far as it has been worked out to date, but state that it is still being used with caution. They have given it to five children. In each case an increase was produced in lead output in the urine varying between tenfold and twentyfold, with a rapid amelioration of symptoms and of many abnormal signs. Byers and Maloof point out that after treatment with edathamil, lead remains in the skeleton, whence it is slowly excreted. Metabolic stress (fever, acidosis or alkalosis) increases the rate of release from the skeleton and its transportation to soft tissues, with production of lead poisoning. Such occasions of metabolic stress are therefore indications for treatment with edathamil.

[1] *Science*, October 30, 1953.
[2] *Am. J. Dis. Child.*, May, 1954.

Abstracts from Medical Literature.

PÆDIATRICS.

Umbilical Hernia in Infants.

G. E. WOODS (*Arch. Dis. Child.*, December, 1953) has studied the natural history of umbilical hernia in 283 infants. In this series a frequency of one umbilical hernia in six infants was recorded. The incidence was higher in twins or premature infants, in breech-born infants, in babies of high birth weight, and in those with unusually long umbilical cords. Mild umbilical sepsis predisposed to herniation, while the formation of granulation tissue at the umbilicus prevented it. Conditions giving rise to hypotonicity or to a rise in intraabdominal pressure favoured the development of hernia. In only one case of this series were symptoms attributable to the hernia, and this one appeared to be of the so-called adult type. Spontaneous cure is a very common event. The use of restrictive appliances, such as strapping and trusses, frequently aggravates the condition and delays spontaneous cure. The last 122 umbilical hernias seen at the infant welfare clinic were not treated in any way. In all but three an automatic cure occurred, and in the three outstanding cases the infants are well on the way to a natural cure. The ultimate incidence is very small.

Ano-Rectal Rings in Infancy.

L. E. HARRIS, H. P. F. CORBIN AND J. R. HILL (*Pediatrics*, January, 1954) have investigated the incidence of ano-rectal rings in newborn infants. They state that such rings are formed by partial failure of absorption of the anal membrane. They were found in 13·8% of 1716 consecutive newborn infants and were four times as common in females as in males. The incidence of constipation, colic, blood in the stool, excessive straining, distension, tear of the anal skin and crying with defæcation was the same in a group of 168 infants with ano-rectal rings as it was in a group of 160 infants in whom the ano-rectal ring was absent. The authors are of the opinion that the majority of ano-rectal rings dilate spontaneously and are not usually the cause of the symptoms often ascribed to them. An unusually thick, tight ano-rectal ring was found in the 4356th infant examined; and though it caused no symptoms in the newborn period, it was felt that it was unlikely to dilate spontaneously and digital dilatation was recommended.

"Aureomycin" and Streptococcal Sore Throat.

H. B. HOUSER, G. C. ECKHARDT, E. O. HAHN, F. W. DENNY, L. W. WANNAMAKER AND C. H. RAMMELKAMP (*Pediatrics*, December, 1953) treated with "Aureomycin" 1009 patients suffering from exudative tonsillitis or pharyngitis, which was predominantly streptococcal in ætiology. A control group of 1035 patients received no "Aureomycin". Five patients in the treated group and 20 in the control group developed rheumatic fever within a thirty-five day interval. "Aureomycin" therapy was also found to eliminate the streptococcus from the naso-pharynx in a large percentage of cases and to inhibit the formation of antistreptolysin. Inhibition of antistreptolysin was found to correspond with the eradication of streptococci. It was shown that penicillin therapy was more effective than "Aureomycin" therapy in eradicating streptococci, decreasing antistreptolysin formation and preventing rheumatic fever. Penicillin, therefore, is regarded as the drug of choice.

Hydrocortisone and Infantile Eczema.

V. H. WITTEN, A. B. AMLER, MARION B. SULZBERGER AND A. G. DESANCTIS (*Am. J. Dis. Child.*, March, 1954) report the use of an ointment containing hydrocortisone in the treatment of infantile eczema. Thirty infants were treated by application of a 2·5% hydrocortisone free alcohol ointment to the affected parts on one side of the body. The lesions on the other side were treated with the same ointment base consisting of wool fat, liquid petrolatum and white petrolatum. Favourable response to the hydrocortisone ointment was noted in 18 of these patients. Photographs are included to illustrate the comparison between the treated and control sides. The authors conclude that in the general run of cases the hydrocortisone ointment as used in these studies constitutes the simplest, cleanest and most rapidly effective of all the topical measures they have adopted in the treatment of infantile eczema.

Primary Tuberculosis After Immersion in Sewage-Contaminated Water.

F. J. W. MILLER AND J. P. ANDERSON (*Arch. Dis. Child.*, April, 1954) report two cases of primary tuberculosis occurring in children after immersion in sewage-contaminated water. Five strikingly similar cases have been recorded in Scandinavia. The sequence of events described is for a healthy child to inhale and swallow contaminated water and suffer a gastro-intestinal upset which clears in a few days. After two or three weeks of apparently good health a febrile illness ensues, which can be shown to indicate primary tuberculous infection. Virulent tubercle bacilli have been found to survive many weeks in sewage even after chlorination. The possibility of this method of infection should be recognized and the sequence of events noted.

Retinoblastoma and Intravenous Nitrogen Mustard Therapy.

CARL KUPPER (*Am. J. Ophth.*, December, 1953) describes the use of intravenous nitrogen mustard therapy in the treatment of retinoblastoma. A child, aged six years, presented with a retinoblastoma of the left eye, the right eye having been enucleated at the age of fifteen months for retinoblastoma. The tumour was irradiated, and at the same time a course of intravenous mustard therapy was begun. An amount of 0·4 milligramme of nitrogen mustard per kilogram of body weight was given in divided doses over an eight-day period for a total dose of 9·5 milligrammes. At the end of fifty days the retina was completely reattached and the retinoblastoma had retrogressed. The irradiation was then discontinued, and three weeks later a second course of intravenous mustard therapy was given. Nine months later there was no evidence of recurrence, and the visual acuity was 20/30 less two letters.

Oral Cortisone Treatment of Infantile Eczema.

L. W. HILL (*New England J. Med.*, June 18, 1953) states that to explore the practical possibilities of oral cortisone therapy, 18 infants under the age of three years were treated. Most cases were severe. Potassium chloride, five grains twice a day, was given to all. It was decided at the outset that not more than 75 milligrammes of cortisone a day would be given to any patient, on the assumption that at this level of dosage serious side effects would be unlikely. In addition to the use of cortisone, each patient was treated as he would have been if cortisone had not been used. It is true that infants need and tolerate enormously larger doses in proportion to their weight than adults, but the author does not believe that they tolerate the larger doses (150 to 300 milligrammes) often given to adults. If the eczema is controlled with 75 milligrammes or less, cortisone should be continued at this level; if it is not entirely controlled with that amount, it is the author's opinion that it is best not to try for a complete "cure" with larger doses. The smallest dose should be used that will keep the eczema under moderately good control and will keep the child comfortable. In one case Cushing's syndrome developed while the patient was taking 50 milligrammes a day. In another the patient did well for a large part of the course on 25 milligrammes a day. It is best to start with 75 milligrammes and, as soon as the eczema is controlled, to effect a downward titration with reduction of 12·5 milligrammes each time to determine what daily dose is necessary for the patient. The general condition is excellent whilst the child is taking cortisone. Cortisone is merely an addition to the generally accepted methods of treatment; it in no sense supplements them or changes the allergic status of the child. 'A search for the cause must still be carried out. When cortisone is discontinued, this should not be done abruptly. One should give 25 milligrammes a day for four days and then 25 milligrammes every other day for a week. The treatment with cortisone is symptomatic only, for the purpose of tiding the child over a difficult period.

Neonatal Tetany and Maternal Hyperparathyroidism.

R. L. WALTON (*Pediatrics*, March, 1954) describes the occurrence of persistent tetany in two prematurely born siblings which led to the discovery of primary hyperparathyroidism in the mother. Convulsions and twitchings commenced in the first-born infant on the eighth day of life and continued despite treatment for two weeks; in the second-born tetanic twitching began on the fifth day of life and persisted for about two weeks. Investigation of the mother led to the successful removal of a parathyroid adenoma. The author states that neonatal tetany has been described elsewhere in association

with primary and secondary hyperparathyroidism and with hypoparathyroidism in the mother. The occurrence of severe tetany or of tetany in the breast-fed infant should lead to a search for parathyroid disorder in the mother.

Goitres in Cretins Without Iodine Deficiency.

L. WILKINS, G. W. CLAYTON AND M. BERTHRONG (*Pediatrics*, March, 1954) report a series of four cases of cretinism in which the thyroid gland was present and capable of taking up iodine. The patients were not living in an iodine-deficient area, nor was there any history of excessive intake of goitrogenic substances. In the two older children goitres were present. The authors state that histological examination of such goitres reveals marked hyperplasia with areas resembling adenomata or carcinomata. This type of hypothyroidism tends to be familial. It appears to be due to inability of the thyroid gland to convert iodine into thyroid hormone. In the absence of a goitre this type of hypothyroidism can be diagnosed only by demonstrating a normal uptake of I^{131}.

Physiological Bowing of the Legs.

J. F. HOLT, H. B. LATOURETTE AND E. H. WATSON (*J.A.M.A.*, January 30, 1954) state that it is recognized anthropologically that virtually all infants have some degree of genu-varum deformity that frequently persists into the second or third year of life before normal physiological development converts the mild bowing into an equally mild degree of *genu valgum*. In some instances, the bowing is largely due to normal distribution of adipose tissue, which in infancy tends to be more heavily deposited on the upper surfaces of the thigh and lower part of the leg, but in most infants some degree of actual osseous bowing is present, although they are otherwise apparently healthy and well nourished. The deformity, which is usually recognized in the immediate post-ambulatory period, is generally not due to rickets, osteochondritis or serious developmental fault. It is thought, instead, to be the result of delayed resolution of normal physiological bowing that is present to some extent in all infants. In most instances, the prognosis for complete, spontaneous regression of the bowing is excellent, the legs usually having become straight by the age of four or five years. X-ray examination is imperative to rule out significant metabolic disease, but results of such examination may be misleading. Particular attention is directed to the confusing mechanical structural changes and exaggerated anatomical variants that are the result, and not the cause, of the bowing.

ORTHOPÆDIC SURGERY.

Congenital Arterio-Venous Fistulæ of the Hand.

RAYMOND M. CURTIS (*J. Bone & Joint Surg.*, October, 1953) states that multiple arterio-venous communications in the hand usually have their origin in congenital remnants of the embryonic *Anlage* of the arteries and veins. However, such abnormal communications may also develop from the normal arterio-venous communications which occur in the hand and are known as the neuromyo-arterial glomus. In this event, this structure apparently becomes uncontrolled as a result of damage. In one of the three cases reported by the author, arterio-venous fistulæ developed in a finger after a burn became infected. He states that this may be an example of the latter type of pathogenesis. The diagnosis of congenital arterio-venous fistulæ is best made by the arteriogram, since it shows the site of the abnormal communications. The operative procedures for the eradication of these minute anastomotic channels must accomplish this end with the least possible sacrifice of the function of the hand. The excision must be staged in some instances, to allow adequate collateral circulation to develop between stages. Excision of the skin overlying the lesions, with replacement by pedicle or other types of skin graft, may be necessary in order to excise all communications. The operations are best carried out under tourniquet. A post-operative arteriogram provides the best evidence of adequate excision. There was no indication in the three cases reported that surgical intervention caused the process to extend into areas where a normal vascular tree existed. One patient has been followed for four years without evidence of recurrence. Excruciating pain in an area of ulceration was a constant finding in the three cases, and this was not relieved until healing had occurred. The author states that although it may not be possible to eradicate every one of the tiny arterio-venous communications in a given patient, in most instances in which the process is localized they can be eradicated sufficiently to allow healing to take place and to prevent progressive gangrene and loss of function of the hand. It is of the utmost importance to recognize the clinical entity early and to institute radical surgical treatment before the process causes irreversible changes in the extremity. In one of the cases reported such an irreversible change had taken place, and amputation of the involved finger was necessary. In the other two cases, adequate excision preserved the remaining hand function.

Chemotherapy of Bone and Joint Tuberculosis.

M. C. WILKINSON (*J. Bone & Joint Surg.*, February, 1954) states that streptomycin and iso-nicotinic acid hydrazide are two powerful drugs lethal to tubercle bacilli when access to the infected tissues is free. For early disease, before ischæmia and necrosis become established, they are curative: afterwards they are not. Constitutional treatment is essential in addition to efforts to exterminate the tubercle bacilli with surgery and antibiotics. The effects of streptomycin on tuberculous tissue were greater after ninety days' treatment than after shorter periods of treatment; the synovial tissues were most affected. Even when specific changes had disappeared after ninety days' streptomycin treatment, gross inflammatory changes remained and no doubt prejudiced full natural healing. The operation of partial capsulectomy and synovectomy may therefore still improve the patient's condition whether specific changes remain or not. Certain patients operated upon late in treatment showed dense fibroid changes in the synovial membrane, which was adherent, as a thickened sheet, to the periphery of the joint cartilages. The gross inflammatory changes found at operation often belied the improved clinical appearance after streptomycin treatment. The duration of treatment for active tuberculosis of the hip or knee requiring displacement osteotomy or fusion has been diminished by the introduction of streptomycin. The stabilization operation can be performed earlier owing to the security from miliary dissemination and secondary septic infection; tuberculous tissues operated upon also heal quicker. Nevertheless, the duration of treatment cannot be reduced indefinitely, because, though the local lesion may do very well, the prognosis depends as much on the incidence of associated tuberculous lesions elsewhere. Because it is difficult to cure patients with tuberculosis of the thoracic part of the spine, and because sound healing seldom occurs in the presence of a persistent perispinal abscess, the author used streptomycin as a safeguard to enable him to operate on 28 patients with persistent spinal granuloma or abscess. The operation with antibiotic therapy seemed the shortest way of arresting the progress of the disease. Three main elements are to be considered in tuberculosis of the hip: the subchondral bone, the synovial membrane and the capsule. Mostly the vitality of the subchondral bone is the crucial factor. In hip tuberculosis of childhood cartilage survives longer than bone. It seemed for these reasons that by partial capsulectomy and synovectomy tissues might be removed in which the circulation was defective, that after such operation revascularization might occur in reformed tissue kept healthy by streptomycin, and that the drug might then obtain access to the subchondral bone, on which the integrity of the joint depended. Of 26 children's hips operated upon the function of only four has been lost, but the same treatment applied to four adult hips has failed. The author points out that before antibiotics were introduced many children recovered from tuberculosis of the knee with a good range of movement. A small number of adults also recovered with useful range of movement, but relapse followed. Partial synovectomy and capsulectomy as an adjuvant to treatment seemed therefore less necessary, especially in children; constitutional treatment and antibiotics might be sufficient to tip the balance in their favour. For adults, however, to ensure permanent recovery of function and to reduce the duration of treatment, every possible therapeutic device seemed desirable. The disadvantage of partial synovectomy in the treatment of tuberculosis of the knee is the impairment of movement after operation. Yet movement gradually returns, and most patients in the author's series have regained movement up to 90°. At operation no attempt is made to perform total synovectomy; only synovial membrane accessible from an anterior incision is removed. Of 16 patients treated so far 13 are well and have good function.

Medical Societies.

AUSTRALIAN PÆDIATRIC ASSOCIATION.

THE fourth annual meeting of the Australian Pædiatric Association was held at Canberra from April 8 to 11, 1954. DR. P. A. EARNSHAW, the President, occupied the chair, and 49 members were present. The Association entertained as official guests the Minister for Health (Sir Earle Page), officers of the Commonwealth Department of Health, members of the staffs of the Australian National University and the Institute of Anatomy and the Editor of THE MEDICAL JOURNAL OF AUSTRALIA. Sixteen private guests attended the meeting.

Annual Report and Financial Statement.

The annual report and financial statement for the preceding twelve months were received and adopted.

Election of Office-Bearers.

The following office-bearers were elected for the year 1954-1955:

President: Professor Lorimer Dods.

Vice-President: Dr. Kate Campbell.

Honorary Secretary-Treasurer: Dr. D. G. Hamilton.

Executive Committee: Dr. P. A. Earnshaw (Past President, Queensland), Dr. R. Wall (Tasmania), Dr. M. Cockburn (South Australia), Dr. R. Crisp (Western Australia).

Election of Members.

The following were elected by ballot to membership of the Association: Dr. Freda Plarre (Victoria), Dr. Alan Williams (Victoria).

Fifth Annual Meeting.

It was decided that the fifth annual meeting should be held at Canberra in April, 1955.

The Pædiatric Approach to Congenital Heart Lesions.

A round-table discussion took place on the pædiatric approach to congenital heart lesions, DR. MOSTYN L. POWELL (Melbourne) in the chair.

DR. CHARLES RICHARDSON (Ballarat) read a paper entitled "Congenital Heart Disease as Seen in Country Practice" (see page 317).

DR. KATE CAMPBELL (Melbourne) read a paper entitled "Congenital Cardiac Conditions in the Newborn" (see page 318).

DR. RUSSELL HOWARD (Melbourne) read a paper entitled "Congenital Heart Lesions: The Surgeon's Viewpoint" (see page 321).

DR. DOUGLAS STUCKEY (Sydney), in opening the discussion, supported Dr. Richardson's remarks about the patient with patent *ductus arteriosus* presenting with symptoms in infancy being often overlooked. Of 76 patients with this condition seen at the Congenital Heart Disease Clinic at the Royal Alexandra Hospital for Children in recent years, only two were under two years of age; yet during the same period of time eight children in this age group with a patent *ductus arteriosus* had died in the hospital. It would seem that the condition was not being recognized at that early age by general practitioners and others.

Dr. Stuckey also supported Dr. Kate Campbell's remarks about the different pattern of congenital heart lesions in infancy. For instance, in childhood and adolescence cyanotic forms accounted for about one-fifth of all cases seen; in infancy the cyanosed child accounted for almost one-half of all cases of congenital heart disease, the increase being due to a greater incidence of the more serious forms such as transposition of the great vessels, tricuspid atresia, pulmonary atresia and the infantile form of coarctation of the aorta.

Dr. Stuckey went on to speak of the manifestations of congenital heart lesions in later life. The patient with coarctation of the aorta might be seen with severe hypertension in adult life when surgery was difficult or impossible. The patient with aortic stenosis might be severely handicapped by *angina pectoris*, and this symptom was prominent in about 20% of cases. Of all the congenital heart lesions, atrial septal defect was likely to remain unsuspected throughout childhood, and might be discovered for the first time in a woman during pregnancy. An unsuspected atrial septal defect of moderate size had been found at post-mortem examination in a man who had died at the age of eighty-five years. Ventricular septal defect presented in two main forms. The classical form of Roger's disease with a small defect and a loud murmur and thrill had a relatively good prognosis. Patients with the other form, a large defect permitting a tremendous shunt of blood from the left to the right side of the heart, usually died in infancy or childhood.

In conclusion, Dr. Stuckey drew attention to the fact that congenital heart lesions now accounted for about one-fifth of all deaths in childhood, many of these in the first six months of life. In the future, surgery would probably be undertaken more frequently in the early years of life, and therefore it was important that all patients should be referred for a competent opinion when the diagnosis of congenital heart disease was suspected, regardless of age. Special diagnostic tests would be necessary when the diagnosis was in doubt and there was a possibility of surgical intervention. A reasonably optimistic attitude should be assumed towards those to whom surgery could not be offered; in particular the normal activities of the child should not be restricted unless there was very good cause. Many congenital heart lesions were symptomless, and these patients needed no restriction. The child who had symptoms soon learned to limit his activities to the point of avoiding discomfort.

DR. T. Y. NELSON (Sydney) said that he thought one of the main purposes of the meeting should be educational. The place of the general practitioner in the problem was difficult, but he should be aware of the developments and of the possibilities of benefit being given to his patients. In the present gathering there would be general agreement about the advisability of subjecting patients suffering from patent *ductus arteriosus* to operation, but there were many practitioners who had to be persuaded of the advisability of this operation. He agreed with Dr. Russell Howard about the advisability of operating on these patients in the first year if cardiac enlargement was present, but would not like to accept the general statement that a child with a patent ductus should be subjected to operation as soon as it was diagnosed. He could see no great advantage in operating in uncomplicated cases before the age of two years. In the group of cardiac defects which presented with serious symptoms in the neonatal period he thought that pulmonary stenosis presented the greatest challenge, as Roth had shown that it was possible to operate successfully on a child of twenty-eight days. In the group who had survived the neonatal period he regarded the cases of tetralogy of Fallot as the chief problem. He thought these patients should be carried on until later childhood unless they were deteriorating so that better operative results could be achieved. In those cases in which a cardiac defect was discovered in later childhood and the child had not suffered much disability, one of the important problems was pulmonary stenosis, and in these cases detailed investigation in a cardiac clinic was the only means of assessing the need for operation.

DR. L. STOKES (Melbourne) spoke of the harm that had been done by unwise Press publicity. Even after operation a child with the tetralogy of Fallot had an abnormal heart. He disagreed with Dr. Nelson over waiting for deterioration before advising operation, and said that many children on whom the operation had been performed at the age of seven and in whom there had been no prior degeneration had justified the decision by their subsequent progress.

PROFESSOR LORIMER DODS (Sydney) questioned the significance of maternal nutritional deficiency as a possible cause of congenital cardiac defects or of any other congenital malformation of the human infant. He was aware of the various forms of congenital cardiac defects—more particularly anomalies of the great vessels—which had been produced in the offspring of rats and other animals by certain forms of nutritional deprivation during pregnancy, but he did not know of any shred of evidence that such defects had occurred in human infants as a result of even the most severe maternal dietary deficiencies.

In 1945 Clement Smith had studied a large group of infants born to mothers in Holland who had suffered six months or more of hunger and semi-starvation during their pregnancies, and had been surprised to find that there was no significant increase in the incidence of congenital malformations amongst the infants.

It was well known that congenital cardiac defects might be genetically determined and that such defects might also follow rubella and possibly certain other noxious influences operating during the early months of pregnancy, but there were obviously very many wide gaps in their knowledge of

the problem. Why should patency of the *ductus arteriosus*, which was relatively rare amongst Mongols, be by far the most common cardiac defect to follow maternal rubella? Why should cyanotic congenital heart disease be very rare as a sequel of maternal rubella? Why should patency of the *ductus arteriosus* be the most common form of inherited cardiac defect and why should septal defects be the most common form of congenital cardiac malformation associated with Mongolism? What other noxious influences apart from rubella occurring during pregnancy might be responsible for the development of congenital cardiac defects?

Professor Dods hoped that some day in the relatively near future it would be possible to arrange very wide scale pre-natal studies of a prospective type which might help to answer the question raised by Dr. Richardson and Dr. Campbell about the part played by maternal nutritional factors in the ætiology of congenital cardiac defects and might help to establish or deny the significance of various known and unknown noxious influences as causes of congenital cardiac malformations.

DR. C. ISBISTER (Sydney) asked what symptoms or signs did the babies whose lesions only declared their presence later show during the first week or so while they were still in the maternity hospital. She felt there must be many adaptive processes, at present unknown, taking place in the baby's body at birth. She had seen many newborn babies who had tachycardia or changing murmurs, or who were difficult to manage, and yet were not eventually considered to have congenital disorders of the heart. She asked if an attempt should be made to discover more about the details of delivery and resuscitation in every case. Many resuscitative measures at present applied by nurses were, she felt sure, unsuitable and possibly dangerous.

Dr. K. Campbell, in reply to Dr. Isbister, agreed with the necessity of knowing all details of delivery and resuscitation. Anoxia at birth caused tachycardia which might last for several days. Further, the *foramen ovale* remained open for a long period. Some authorities held that closure of the *ductus arteriosus* was due to falling systolic blood pressure and to the tone of the vessel wall, but she wondered why it closed when accompanying the tetralogy of Fallot. She thought there must be some malformation of the ductus itself and asked what was the structure of an abnormal ductus.

DR. R. SOUTHBY (Melbourne) asked whether there was any relation between the intensity of the murmur and the size of the ductus in early life, and also what were the risks of not operating on a patent *ductus arteriosus*. Were there any statistics about the incidence of subacute bacterial endocarditis?

Dr. R. Howard replied that he did not know the statistics in that respect, but that although parents often claimed that affected children were quite well before operation, after the operation they realized they had been wrong and gave many instances of improved ability and activity. In point of fact these children were not normal and their hearts were labouring. He was sure elasticity of the ductus varied directly with the child's age, so that it was easier to operate on the ductus in younger children. However, the diagnosis was difficult in children of less than one year old and, as accurate diagnosis was essential before operating, this meant that in practice operation was rarely possible before the age of three or four years.

DR. E. S. STUCKEY (Sydney) said that he wished to refer to three small points: (i) Dr. Southby had suggested that the patent ductus discovered by accident need not be operated on in early life. Surely if one considered the quantity of blood being shunted unnecessarily from the aorta back to the left side of the heart it must be a good thing to have this corrected as easily as possible. He wished to support the statement by Dr. Howard that operation was easier in quite young children. (ii) With regard to tetralogy of Fallot, however, he felt that there was reason for waiting if the child could be nursed through the early years, for the reason that the small size of the great vessels made a satisfactory anastomosis difficult in the smaller children. (iii) Finally he referred to one hazard in the operative cure of patent ductus, and that was cardiac arrest under anæsthesia, apparently preceded by supraventricular paroxysmal tachycardia. He had had this happen twice. In one case cardiac massage restored the beat, but the ductus had not been tied. Some months later all concerned had a worrying time when this child was submitted to second operation. In the other case the beat was also restored by cardiac massage, and they went ahead and tied the ductus with a successful outcome. He wished to ask Dr. Howard if he had had any similar cases. He had begun to give the ductus patients quinidine before operation as a precautionary measure.

Dr. R. Howard replied that his practice was to use digitalis in a similar way.

DR. FELIX ARDEN (Brisbane) agreed that the first task of the pædiatrician was to select patients suitable for surgery and send them to the right person at the right time; yet his more frequent lot was to give advice about children whose heart lesions were inoperable. It was his duty in these circumstances to secure for the child the fullest happiness and for his parents peace of mind. All too often they had been given the impression that if they were lucky an operation could be done, implying that inoperable lesions such as atrial or ventricular septal defects were a misfortune. They understood the child's heart was "damaged" and concluded that he must never exert himself. If the doctor was certain that the heart lesion could not be altered by surgical means he should try to minimize it in the patients' minds. This meant the adoption of a cheerful and optimistic attitude about it. Members had just heard how a large atrial septal defect was discovered in an octogenarian. It also meant allowing full activity, limited only by the child's inclinations, and avoiding frequent visits to the surgery and constant listening to the murmur. Remembering that the myocardium was not diseased and that it responded to exercise or disuse in the same way as normal heart muscle, the pædiatrician should have the courage to send these children away to lead a normal life, rather than take steps which suggested invalidism.

Dr. Arden thought that the risk of endocarditis was very small. In the previous five years he had seen only two instances of acute bacterial endocarditis and no cases of the subacute variety.

DR. JOHN COLEBATCH (Melbourne) said that from his experience in a fortunate association with Dr. Powell at the Royal Children's Hospital and with Dr. Campbell at the Melbourne Women's Hospital, he offered a few comments concerning the newborn with congenital heart disease. In the diagnosis of cases without definite cyanosis or syncopal attacks the history given by the nurse was of the greatest help. Tachypnœa and tachycardia were very difficult to evaluate in the first week, when the range of normal figures was so very wide. A good description of the feedings, however, was the commonest easily obtained guide to diagnosis—the baby did not suck well, or was slow with the feedings, or they tired and distressed him, or he was unable to take an adequate feeding, or he took it with an effort and shortly afterwards vomited or became blue. A good nursing record could certainly simplify diagnosis.

Dr. Colebatch did not think it was possible to overemphasize the trap that cardiac bruits provided in the first week. Some of the loudest bruits he had heard during the first three or four days had been associated with moderate or severe atelectasis and within a week or two they had disappeared completely. Ventricular septal defect and a few other anomalies might, of course, produce an audible bruit at that stage. Frequently, however—one might almost say usually—the baby with congenital heart disease had no audible bruit in the neonatal period, at least not in the first week or so. In the previous year a registrar with a lot of experience had come to Dr. Colebatch to discuss a baby of two days who was cyanosed and not doing well. Dr. Colebatch asked if he had a congenital heart lesion. The reply was: "I don't think so, sir. I can hear a murmur."

Dr. Powell had raised the question of disposal. Dr. Colebatch would like to ask at what stage should a baby who was thought to have a congenital lesion be referred to the cardiac clinic for screening, angiography or other special investigation. He thought that after a presumptive diagnosis had been made in the neonatal period, pædiatricians sometimes made a mistake in keeping such a baby languishing in a hospital cot until further investigations were all done. Perhaps the chief need during this time was sound mothercraft nursing in a good babies' home, or in its own home if circumstances permitted.

Finally, with regard to the possible ætiology of these anomalies, like Dr. Campbell, Dr. Colebatch was very interested in Warkany's malnutrition theory and very keen that the diet should be carefully investigated in every case. In support of Professor Dods, however, he believed that the case for malnutrition had yet to be proved in human beings, and that they should keep an open but inquiring mind about it. Dr. Colebatch recalled the classic story of Clement Smith, who visited Holland at the end of the war to study the effects of wartime under-nutrition there. On returning to America he had to tell Warkany that although sterility had increased in Holland, there was no increase of congenital malformations. Warkany had at first been puzzled, and then the light had shone in his eyes as he said: "Still, could any congenital malformation happen to a person more serious than not to be conceived at all!"

DR. R. GREEN (Sydney) asked if Dr. Campbell knew whether the incidence of congenital heart lesions had increased.

Dr. K. Campbell replied that she thought that any increase was more apparent than real. She wondered if there could be any possible transmitted relationship between mothers whose pre-conception diet had been poor, and babies who showed certain abnormal patterns of bowel function or who were nutritional problems in early life. There were certain points in common.

DR. V. L. COLLINS (Melbourne) said that he thought most of them would agree that Dr. Felix Arden's statement was one of the most important made that day. There was no doubt that with all the modern technical diagnostic aids there was a tendency to forget the management of the patient as a person. Surely when advising a parent they should take advantage of the statistical fact that certain patients with congenital heart disease might live to a normal age, and as often as possible reassure them that their child should have the opportunity to do all the things that a normal child was permitted to attempt. A few years previously Dr. Collins had attended a cardiological clinic in another country. A nervous, anxious mother was telling the physician what she did for her blue baby, aged eighteen months. She was afraid he might again suffer from bronchopneumonia; she tried to prevent him doing anything that might make him bluer; she did nothing that might make him cry; the parents refrained from playing with him because they thought the increased cyanosis from his laughter might harm him. All the physician told this distraught mother was that when her child was two years old she would have further investigations done to see if an operation was possible. She was given no further advice, and yet her great need was to be given an explanation of how to live with the baby, how to enjoy him in spite of his blueness, and to permit and encourage him to do all the things that a baby of his age could do. For the same reason Dr. Collins would agree with Dr. Stokes that there must be a place for Blalock's operation in Fallot's cases. The operation could change the child's life, even though considerable disability remained. Dr. Collins had seen this well exemplified following, he thought, the third Blalock operation in Melbourne. The girl had lost all cyanosis after operation and so she was no longer teased by the other children because of her blueness. She lost the constant muscular pains which had previously been present. She could ride a bicycle. These things made an enormous difference to her whole outlook and social relations and in Dr. Collins's opinion clearly justified such an operation.

DR. C. LEE (Sydney) asked Dr. Howard what were the late results of early operation on the patent *ductus arteriosus*.

Dr. R. Howard replied that it was as yet too early to say, but the results so far had been very good.

DR. A. WILLIAMS (Melbourne) had been interested in observing microscopic changes in the pulmonary arterioles which had been evident in some sections he had examined. He wondered whether delay in closure might be due to abnormalities of pulmonary vascularity.

DR. N. WETTENHALL (Melbourne) referred to the diagnosis of congenital heart disease. Many patients were referred because of a functional bruit, the characteristics of which were well known to pædiatricians. One particular bruit, however, was worthy of special mention, and this was the venous hum, which did not appear to be well recognized. Its character was not unlike that of a patent ductus, though it was often audible on the right-hand side of the sternum as well as on the left. Moreover, it varied greatly with alteration in position. The diagnostic characteristic of such a bruit was that it could be much reduced or even obliterated by pressure on the jugular vein.

Dr. Wettenhall also raised the point that some children with coarctation of the aorta were regarded as suffering from rheumatic fever because they presented with pains in the legs, and as a result of the finding of a systolic bruit, they were kept in bed for many months. This was a diagnostic error which should not occur, as palpation of the femoral arteries always revealed absent pulses. This sign was of such diagnostic value that coarctation of the aorta could be recognized in the youngest infants.

DR. J. PERRY (Melbourne) said that he did not know of any statistics showing the degree of danger of subacute bacterial endocarditis developing later, but his own findings were only two or perhaps three blood cultures positive for *Streptococcus viridans* over a six-year period.

DR. S. STENING (Sydney) asked Dr. Campbell what advice she would give to mothers of affected children regarding future pregnancies.

Dr. K. Campbell replied that she could only suggest that if after careful investigation of the diet before conception and in early pregnancy no deficiency or abnormality could be found, the mother should be advised to go ahead and have another.

Dr. R. Howard, reverting to Dr. Southby's question about the relation of the intensity of the murmur to the size of the *ductus arteriosus*, said that although there were well-authenticated reports of large ductuses which had been unaccompanied by a murmur, his own experience was that the relation was a direct one. However, he thought there was a more basic characteristic than intensity which he could only describe as an auditory impression of much blood going through the ductus.

Dr. M. Powell, the chairman, in summing up, made the following points.

1. There were three main groups of cases which presented for diagnosis. The first group was a pathetic group of infants with a critical survival period ending at about six months of life or before. In these few months were seen the slow deterioration and final episode of the severe, uncompensated defects such as gross pure pulmonary stenosis, the extreme tetralogy of Fallot with pulmonary atresia, transposition of the great vessels with inadequate compensating shunts, huge septal defects and the anomalous origin of the left coronary artery from the pulmonary trunk. This was the group which was identified in the first few days of life as desperately ill, distressed, dyspnœic, pallid or cyanotic babies in which cardiac rates were rapid and bruits inconstantly heard, and in which radiology demonstrated occasionally a classical pulmonary stenotic lesion, with oligæmic lung fields, but more often a nondescript ballooned contour of a heart in its last stages. Management could be directed only to control of the great feeding difficulty in the face of persistent dyspnœa, the regime of frequent small-volume feeds, and the rather face-saving administration of oxygen which all too often could not be absorbed by the ischæmic lung fields. Accurate diagnosis was very difficult, and the hazards of operative interference were great at this early age. Nevertheless, there should always be an urge towards correct diagnosis. Such methods as the hypothermic technique might revolutionize the surgical approach. Already, a transposition of great vessels had been corrected in the newborn period with this method which so slowed the heart rate and diminished oxygen requirements that clamping of the two *venæ cavæ* for upwards of five to ten minutes could be achieved. This group would require intensive study, and in a proportion—perhaps a small proportion—some alleviation might be possible. A second group had patients who, though often moderately distressed and cyanotic, were progressing fairly well at the critical six months period. These were the cases which gave time and opportunity for more accurate assessment, for catheter and angiographic studies, and, if surgery was to be attempted, a rather larger heart and great vessels with which to grapple. In this group were the less severe pulmonary stenotic lesions—those, for example, in which the right ventricle was eased of some stress by a septal defect, the tetralogy of Fallot, the fairly large septal defects, transposition of the great vessels with good compensating shunts, the Eisenmenger syndrome, and the anomalous pulmonary venous return to the right auricle. Dr. Powell endeavoured to allow a normal life, short of competitive sport, to those patients and to offer vocational advice. Finally, there was the big group of patients whose symptoms were absent or minimal, but in whom a murmur was heard whilst they were seeking advice for some unrelated condition or was heard during routine medical examination. Here they had the patent ductus, the coarctation of the aorta, the ventricular or auricular septal defect with minimal shunt, or cases of aortic and pulmonary stenosis with definite signs but few symptoms. In this group, also, were the patients with innocent bruits.

2. Basically, what was required was a diagnosis upon which to plan a programme for the child, whether it was of an operative nature, a period of observation, or an instruction to carry on a normal life and completely forget the heart altogether.

3. It would appear that the best approach to a diagnosis should be by the use of a clinic dealing with large numbers of patients, for it had to be the concentrated experience gained therein which would provide the most accurate means towards correct diagnosis.

4. In regard to diagnosis, clinical methods were still of vast importance, and would provide a very accurate diagnosis in

a high proportion of cases, the patent ductus being, of course, a prime example of this.

5. Discussing the ætiology, Dr. Powell said that they had found maternal rubella in very few of the cases at the Children's Hospital Clinic, to wit, about six in four hundred congenitally affected hearts. It would appear that the nutritional factors mentioned by several of the speakers might well play a preponderant part.

6. Regarding the question of the diagnosis of the severe infant type of patent ductus, Dr. Powell said that the infants had presented with pallor, dyspnœa, failure to thrive, very large hearts, and a group of bruits, which were as follows: (*a*) a long harsh systolic bruit maximum in the second left interspace coupled with a very loud clicking split-second sound in that area; (*b*) in some of the cases a definite diastolic bruit to the left of the sternum which probably represented the Graham-Steele murmur of pulmonary incompetence; (*c*) at the apex quite a loud systolic and mid-diastolic bruit quite reminiscent of the bruits in rheumatic carditis. The true continuous bruit of the classical patent ductus was missing in most of the cases, and this was what had rendered the diagnosis difficult. Pulse pressures were high, two patients showing a systolic blood pressure of 120 millimetres of mercury and a diastolic pressure of 0, and this was reflected in the bounding femoral pulses.

7. In regard to Dr. Nelson's query about the necessity for operation at this age, Dr. Powell said that the obvious cardiac embarrassment, and failure to thrive, and even the dysphagia produced by the cardiac enlargement, left no doubt that operation was indicated. In regard to advice to parents of children with patent ductus in which there were no obvious symptoms, the approach was to inform the parents that doctors were not thinking of the present when they talked of operation, but rather of 15 to 20 years later.

8. Regarding the question of Dr. Southby's, the main complication of the patent ductus was not subacute bacterial endocarditis but slow progressive cardiac failure. Actually, in 110 cases of patent ductus, they had had one case of subacute bacterial endocarditis.

9. The optimistic approach stressed by Dr. Collins and Dr. Arden was always advisable when possible, but, of course, there were cases such as those of auricular septal defect in which at an early age cardiac enlargement and pulmonary congestion indicated a poor future. Still, it was always possible to say that with improved knowledge and increased operative skill, the child's condition might be ameliorated in this way at a later date. This gave the straw to which the parents could clutch as the years went by.

10. Dr. Wettenhall also had asked about the frequency of functional bruits: 54 bruits of this type had been present in a recent series of 457 cases at the Royal Children's Hospital. These bruits were Grade I in intensity—they were faint, they were very short, and they either honked, squeaked or twanged. The venous hum which sometimes caused difficulty was always heard better above the clavicle than below it, and could be altered or abolished by compression of the venous channels in the neck.

11. Finally, the title of the session was "The Pædiatric Approach to Congenital Heart Lesions", and it did seem that that approach, especially in its surgical aspects, had always to be in the confines of the pædiatric age group.

Congenital Bladder Neck Obstruction.

DR. E. S. STUCKEY (Sydney) read a paper entitled "Congenital Bladder Neck Obstruction" (see page 321).

The paper was discussed by DR. F. D. STEPHENS (Melbourne), who illustrated his remarks by a series of lantern slides.

Subacute Pulmonary Collapse with Pyogenic Infection.

DR. HOWARD WILLIAMS (Melbourne) read a paper entitled "Subacute Pulmonary Collapse with Pyogenic Infection in Children: Its Relationship to Permanent and Reversible Bronchiectasis". He said that acute pulmonary collapse associated with infection occurred relatively frequently in infants and children, and that the collapsed lobe or segment usually expanded as the infection resolved. Failure of the lung to reexpand could be due to several causes; the most common was persistence of infection, while continued bronchial obstruction, defective breathing or destruction of lung tissue was less common. If the lung remained collapsed for longer than four weeks the lesion could be arbitrarily described as entering a subacute phase and permanent collapse and bronchiectasis were then likely to occur.

Dr. Williams said that the object of the paper was threefold. First he wanted to clarify the clinical syndrome of subacute pyogenic pulmonary collapse so that the clinician could readily make a diagnosis. Second, he wanted to demonstrate that bronchiectasis was present in the collapsed infected lobe, and that with satisfactory treatment complete or partial resolution was possible in many patients. Third, he wished to discuss the importance of this type of chest lesion as an ætiological factor in bronchiectasis in children.

Clinical Features.

Dr. Williams then reported thirty-seven patients with subacute infected lobar collapse who had been studied over a six-year period from January, 1948, to December, 1953, in the Clinical Research Unit at the Royal Children's Hospital, Melbourne. The ætiological factor primarily responsible for collapse was non-specific respiratory tract infection in 20 cases, pertussis in nine, measles in two, anæsthesia and operation in two, inhaled foreign body in one and allergic bronchitis in three. Patients with collapse due to tuberculous glands, fibrocystic disease of the pancreas and respiratory muscle paralysis were excluded from this survey.

Pyogenic infection had been present in all patients as evidenced by the coughing or aspiration of mucopus from the bronchial tree. Sixteen of the patients were boys and 21 girls. Their ages ranged from ten months to fourteen years, three were under two years, 22 between two and six and 12 between six and fourteen years.

The common pattern of illness had been that of a healthy child who developed either non-specific respiratory tract infection, pertussis, pneumonia, measles or an attack of allergic bronchitis, but then failed to make a satisfactory recovery. The two clinical features which indicated an infected collapsed lobe were failure to regain normal health and residual cough. The symptoms of ill health reported by the mother were usually listlessness, refusal to play, irritability, poor appetite and either loss of, or failure to gain, weight. Cough was present in all patients, but frequently was not troublesome. Sputum was not spontaneously coughed up by any patient, although all had infective exudate in the bronchial tree.

Clinical examination had usually revealed a child who was not well, was often pale and had lost weight. Frequently posture and muscle tone were poor, the child being round shouldered with thoracic kyphosis. Upper respiratory tract infection was uncommon; two patients had intermittent infection and two allergic rhinitis. There had been two constant physical signs—inspiratory crepitations and diminished air entry over the affected lobe or lobes. These signs could frequently be elicited only when the child coughed and breathed in deeply. Other signs such as impaired percussion note, tubular breathing or diminished movement of the affected side of the chest were frequently absent. Almost all children could be taught to cough mucopus by posturing the child over the examiner's knee, percussing his back and telling him to "cough and spit it out". Special training was necessary in the very young child. The sputum had almost invariably been in the form of yellow discrete blobs, but occasionally thin or thick streaks of mucopus were produced. The sputum was cultured in 33 instances and yielded the following organisms: *Staphylococcus aureus*, 9; hæmolytic streptococcus, 5; *Streptococcus pneumoniæ*, 3; *Hæmophilus influenzæ* type B, 2; *Bacterium coli*, 1. In three of the patients with a culture of *Staphylococcus aureus* there was an associated organism; *Streptococcus pneumoniæ* occurred in two and β hæmolytic streptococcus in one. No growth was obtained from culture of the sputum in 13 cases.

The lobes affected were always those draining against gravity. In 27 patients the lesion was unilateral, in 10 bilateral, while the right side was affected in 17 patients, the left side in 10. A combination of right middle and right lower lobes was the commonest lesion and occurred in 11 patients. The right lower lobe was involved alone in five patients and the left lower lobe in eight.

Bronchoscopy.

Bronchoscopic examination in all patients revealed mucopus coming from the affected lobe bronchus, but in no case was the bronchial lumen plugged with thick tenacious mucopus or the lumen occluded by inflammatory swelling. In the larger number some inflammatory changes were observed in the mucosa of the bronchus of the affected lobe or lobes. A bronchogram in every case demonstrated collapse and bronchiectasis of the affected lobe or lobes. The remainder of the bronchial tree was normal.

Treatment.

Dr. Williams said that there were three principles of treatment: rest to improve general health, chemotherapy to assist resolution of the lung infection, and physiotherapy to promote reexpansion of the collapsed lung. The regime followed was usually bed rest in hospital until the general health improved and weight was gained. Chemotherapy was determined by two considerations, first by the type of organism cultured, and second, by the subacute nature of the lesion. The drugs used were penicillin, or streptomycin or one of the sulphonamides, or a combination of these for approximately two weeks, and then aureomycin or terramycin for a further two to three weeks. Continued chemotherapy for two to three months was necessary for some patients in whom the infection was very slow to resolve. The aim of physiotherapy was to restore to full function all muscles of respiration, especially the diaphragm. Forcible expiration squeezed exudate from the lung into the bronchial tree whence it was coughed, while full inspiration aided pulmonary expansion. Chest percussion assisted coughing and helped expulsion of exudate. Physiotherapy was carried out at two-hourly intervals mainly by the nursing staff who were directed by the physiotherapist. It was only by this means that frequent daily treatment could be maintained.

Results.

Dr. Williams said that of the 37 patients with subacute collapse and bronchiectasis 16 recovered completely, the lobe expanded, the bronchi returned to normal and the child remained well. Thirteen patients partially recovered, the lobe partially or completely expanding, but one or more of the bronchi remained dilated. Eight failed to recover and the lobe remained permanently collapsed and bronchiectatic. (See Figure I.)

An analysis of the main features of each of these groups gave some insight as to the reason for the varied results. In the group with complete resolution or reversible bronchiectasis the two outstanding features were early diagnosis, the lesion being present for periods varying from five to twelve weeks, and adequate treatment. The group of patients with partial resolution of the lung lesion differed from the first group in that many of the patients were younger. Physiotherapy could not be carried out adequately in the very young patients owing to lack of cooperation. Other reasons for incomplete resolution were later diagnosis and inadequate chemotherapy in some instances. In the group of patients in whom the lung remained permanently collapsed and bronchiectatic the two significant features were late diagnosis, the lesion usually having been present for over six months, and little or no treatment.

Dr. Williams in discussing these results said that it was of considerable importance for the family doctor or pædiatrician to diagnose promptly the child with a collapsed infected lobe, for by so doing he would have made an important step in the prevention of permanent collapse and bronchiectasis. The second key to the prevention of permanent lung damage was the early institution of adequate treatment. The group of 16 patients with complete resolution supported this view, while the group of eight patients whose condition failed to resolve and were left with permanent collapse and bronchiectasis clearly showed the unfavourable results of late diagnosis and poor treatment.

Diagnosis was essentially clinical and was not difficult. If the physician realized that, any child who had not promptly recovered his well-being and had a cough after non-specific respiratory tract infection, pneumonia, pertussis or measles should be suspected of having an infected collapsed lobe. In such a case the physician should specifically seek for crepitations, diminished air entry or distant bronchial breathing over one of the lobes draining against gravity. Posturing and chest percussion to produce mucopus were essential to ascertain the presence of exudate in the bronchial tree. A tentative clinical diagnosis of pulmonary collapse and infection could be made in a child with such a clinical history and examination. Radiological examination of the chest and culture of the sputum were necessary to complete the diagnosis.

Consideration of the main factors in each of the three groups of patients revealed the importance of early diagnosis and adequate treatment. Even with early diagnosis, several factors made it difficult to obtain complete resolution. The first was the amount of lung destruction already present and the second was the degree of cooperation of the patient with physiotherapy. The only control the clinician had over the former was early diagnosis. Age largely determined the second, for in a child under three years cooperation with breathing and posture was difficult. Vigorous laughing or crying probably did more than any other measure to restore respiratory function in such a young child.

Finally, Dr. Williams asked whether this syndrome of subacute pyogenic infection and collapse was an important cause of bronchiectasis. In other words, did lobar collapse and infection of lung tissue precede the development of bronchiectasis in many or all cases? He said that that question could be answered only indirectly, for there were no clinical reports of the development of bronchiectasis from the initial stages in large numbers of children. The clinical picture in the group of 37 patients studied was characterized by certain features. The condition developed in a well child

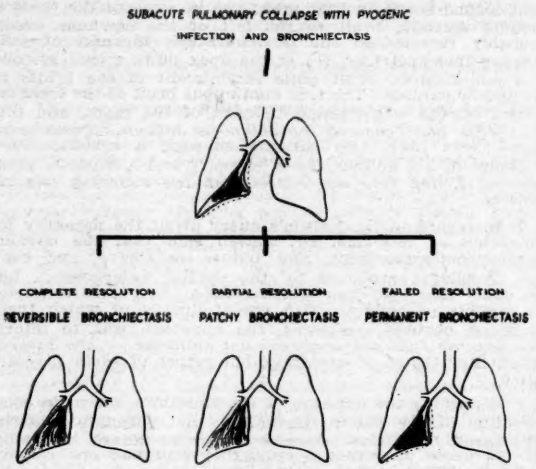

FIGURE I

with a clearly defined respiratory tract infection. The lesion was frequently unilateral and most often right sided. Upper respiratory tract infection was noted in only two patients. Only three children developed the lesion under the age of two years. The fully developed lesion was frequently symptomless or symptoms were minimal. Thus there was a definite resemblance to the group of patients who developed bronchiectasis after invasion of the bronchus with tuberculous lymph nodes.

Using criteria of the pattern of bronchiectasis that occurred in children with subacute collapse and pyogenic infection and applying them to a group of 300 consecutive cases of bronchiectasis at the Royal Children's Hospital, Dr. Williams thought that 20% could be reasonably attributed as being due to this mechanism. It was fully realized that this figure was an approximate one, for bronchiectasis was usually diagnosed several years after its inception, and there could be errors in the reconstruction of the mode of development of bronchiectasis from a clinical history and the pathological nature of the long-established lesion.

Summary.

In summarizing his remarks, Dr. Williams said that:

1. The outstanding clinical features of the 37 patients with subacute pulmonary collapse and pyogenic infection had been failure to regain health and a residual cough after an acute respiratory tract infection, pertussis or measles. The clinical signs were crepitations and diminished air entry over one or more of the lobes draining against gravity and the coughing of mucopus.

2. Dilatation of the bronchi in the collapsed lobe or lobes had been found in all patients. The collapse and bronchiectasis had been shown to be capable of complete resolution in sixteen patients whose condition was diagnosed early and who had adequate therapy. Thirteen patients had achieved only partial resolution; inadequate physiotherapy on account of the very young age and slightly later diagnosis were the factors responsible for these results. In contrast late diagnosis and poor treatment in eight patients had resulted in permanent collapse and bronchiectasis in all.

3. The type of bronchiectasis following subacute collapse with pyogenic infection had certain distinguishing features,

namely, that it was frequently unilateral, often unilobar, often dry and rarely associated with upper respiratory tract infection.

4. This type of lesion was not a common cause of bronchiectasis in children. It possibly accounted for about 20% of a consecutive series of 300 cases.

DR. DOUGLAS GALBRAITH (Melbourne) congratulated Dr. Howard Williams on his paper and especially on the method of presenting the development of bronchiectasis from its beginning, instead of using a retrospective analysis of possible causation in established cases. He asked Dr. Howard Williams if he had found any one organism, or group of organisms, predominating.

Dr. Howard Williams, in reply, said that *Staphylococcus aureus* had been the most common associated organism.

DR. D. G. R. VICKERY (Sydney) said that in Sydney, in connexion with children under three years of age who came to hospital, he had adopted the practice of obtaining somewhat more of their acute history than was obtained in the average case of bronchiectasis. He arranged for them to come back for reexamination by X rays after a month. He had observed that children with small bronchi were likely to suffer from a collapse. In answer to Dr. Galbraith, he said that recently he had read an article in an English journal which showed that in 90% of cases there was an organism known as *Hæmophilus influenzæ*. Whether that was peculiar to England Dr. Vickery did not know, but in Australia *Staphylococcus aureus* was the predominant cause.

Problem of the Deaf Child.

A round-table discussion on the problems of the deaf child was held; DR. J. F. WILLIAMS (Melbourne) occupied the chair. He said that there had been some discussion whether the subject of the deaf child was one which the Association should consider at its annual meeting, but the ill effects on development of partial or complete deafness should leave no doubts on the matter. The choice of a psychiatrist to be chairman also seemed to require some comment, and Dr. Williams's only excuse was that psychiatrists with their particular interests had to face many of the problems associated with deafness, though it was only in comparatively recent years that these problems had received proper attention. In pre-war years the work of the Ewings in Manchester on high tone deafness as the cause of aphasia in children had roused considerable interest, for these cases of congenital word deafness or auditory imperception with apparent preservation of hearing had always been puzzling and fairly common. It was, however, the war-time epidemic of maternal rubella and its effects on the embryo with the resulting wave of deafness in pre-school children that had led to widespread interest amongst pædiatricians and others. The development of hearing aids that could be utilized by children and the possibility of detecting partial hearing defects within the first three years meant that much of the intellectual crippling, speech defects, emotional instability and behaviour problems associated with undetected and uncorrected hearing loss could be avoided. Hopes were roused that all that need be done was to estimate the range of hearing loss, fit a hearing aid that corrected this, and let the child mix normally with its colleagues, but it had soon become apparent that considerable help and supervision were required in addition. The earlier the loss was discovered and treated, the better the prognosis, and various techniques had been developed to enable this early recognition, for example, the peg board, the peep show and more recently the conditioning of the psycho-galvanic response. The last-mentioned promised to enable recognition in the early months of infancy. It remained true, however, that the first suspicion of deafness rested with an experienced observer who was alive to the possibility of this being the cause of retardation or difficulties in development. The observer might well be the parent, the family doctor or the pædiatrician, but it was regrettable that at times it was not until the child reached school age that it was discovered. It was also unfortunately true that at times suspicions of parents had been allayed by optimistic and quite misleading statements by those who should know better. That led to loss of time and a big leeway to be made good, with bad habits and a mental set often strongly averse to the receipt of help, for example, hopelessness, antagonism and at times aggression. Satisfactory testing and later teaching could be made very difficult indeed by such attitudes, and at the best of times it required skill and patience of a very high order.

Deafness resulting from maternal rubella no longer presented as a big problem, but the numbers of children partially or wholly deaf owing to other causes were still very considerable and presented many problems.

The succeeding speakers, Dr. H. O. Lancaster, Dr. V. L. Collins and Dr. F. W. Clements, would deal with some aspects, and Dr. Davey had promised to open the discussion which would, it was hoped, be general.

DR. H. O. LANCASTER (Sydney) read a paper entitled "Deafness due to Rubella" (see page 323).

DR. V. L. COLLINS (Melbourne) read a paper entitled "The Early Recognition of Deafness in Childhood" (see page 324).

DR. F. W. CLEMENTS (Sydney) read a paper entitled "Problems of the Deaf Child: Incidence and Institutions" (see page 328).

The discussion was opened by DR. PATRICIA DAVEY (Sydney), who congratulated the Association on its decision to discuss the subject which was bristling with difficulties. The first problem was one of terminology defining the application of "deaf" (total hearing loss) and the partially hearing and partially deaf.

Into the last-mentioned group fell a great number of children with a moderate loss whose hearing varied considerably, being on some occasions audiometrically normal. Hearing for speech was the major problem in these children, as in all hearing losses. From their defect came heedlessness, inattention, apparent inconsistencies in behaviour, often chastisement at school and at home. Progress at school might be erratic or the moderately hearing handicapped children might be classed as backward. The mother's story was important in its negative aspects as also from her observations. These children could not be adequately or satisfactorily assessed in the absence of the mother.

PROFESSOR LORIMER DODS (Sydney) emphasized the need for complete and careful studies of the ætiology of deafness during infancy and childhood and pointed out that such studies involved a great deal of time and effort. Dr. Lancaster had recently discussed with him the possibility of an ætiological survey of approximately 300 children attending a school for the deaf in Sydney and this had sounded a relatively simple problem, until Dr. Patricia Davey had pointed out that an adequate review of each child and his history would probably occupy at least three hours. Professor Dods felt that studies of this type should be carried out in all institutions responsible for the care of deaf children, and that if these results were combined with some "prospective" pre-natal studies, it should be possible to make some reasonable statement about the ætiology of those forms of deafness which were at present vaguely labelled as "congenital". Professor Dods also stressed the importance of Dr. Lancaster's recognition of the epidemicity of deafness in Australia and New Zealand, a discovery which had prompted a search for the possible epidemicity of other congenital defects. Although that search had so far failed to reveal any epidemicity of other congenital malformations, it represented a line of investigation which was well worth preserving.

DR. ROBERT SOUTHBY (Melbourne) asked whether Dr. Lancaster could say what proportion of rubella children were also blind, and if Dr. Collins could tell them whether there were any means provided for the training of children who were deaf and blind. Those children constituted a special problem.

Dr. Lancaster replied that he had not gone into details because he did not think it was practicable to do so.

DR. V. L. COLLINS (Melbourne) said that the facilities for dealing with those handicapped children were poor. The Blind Institute in Melbourne had offered to take babies who were blind and deaf, but he did not know of any adequate facilities.

DR. HOWARD WILLIAMS (Melbourne) said that he would like to know in what percentage of deaf children a late diagnosis was made, owing to the error of the doctor in recognizing the pattern, and what percentage was due to the failure of the parents to recognize that there was something wrong with the child and not to bring it along until late.

Dr. V. L. Collins said that he had not gone into the percentages; more often the parents were right, although the doctor said he was right. That was where many mistakes were made. The parents had often suspected deafness for some time, but delayed bringing the child along because of the other factors that Dr. Collins had mentioned, such as the child babbling, and so on. On the whole the parents were usually right. Dr. Collins knew of only one instance in this series in which the mother did not suspect deafness, but in which there was a history of maternal rubella and the doctor sent the child along for testing, which proved that the child was severely deaf. That child was two and a half years of age.

DR. FELIX ARDEN (Brisbane) mentioned the case of the partially deaf child whose trouble was due to obstruction of the Eustachian tubes. It was not uncommon to see such children both before and after an operation for removal of tonsils and adenoids and to find that the retraction of the ear-drums and the deafness persisted unless inflation of the tubes had been carried out. He thought it important not to be satisfied with simple removal of the tonsils and adenoids in such instances.

Dr. P. Davey said that the answer to that was that there was a minimum of residual tissue around the Eustachian tube, and that was where radon came in.

DR. KATE CAMPBELL (Melbourne) asked Dr. Collins whether he saw many children suffering from deafness associated with kernicterus.

Dr. V. L. Collins replied that in his series there were none. One group of American figures gave 4% for rubella and 4% for kernicterus, but Dr. Collins could not give the figures exactly. In the previous year there had been none.

Dr. Kate Campbell said that she did not know the exact terminology, but she thought it was supposed to be some spoiling of the central hearing. She was interested in the figures supplied by Dr. Gerrard, of Birmingham. He had dealt with a large number of children and found a high percentage of deafness due to kernicterus.

DR. E. B. SIMS (Adelaide) said that in South Australia the Education Department had appointed an advisory teacher for the hard-of-hearing in 1947 and the services there had developed slowly along the usual lines. The main aim was to establish speech and hearing classes in the ordinary school, so that the ordinary social experiences should be retained. The advisory teacher had told Dr. Sims that there were three big deficiencies in the service. The first was the lack of provision of residential hostels for country children, which was a real need. The second lack was the absence of provision for children with multiple handicaps and, thirdly, a deficiency which developed as the children grew and there was no vocational guidance for their future. There had been no long-range survey to fit the children for their later place in the community. Dr. Sims would like to know what was being done in the other States.

Dr. V. L. Collins said that they still remained problems and it was something which would have to be faced. It had been found that the child who had been taught lip reading and speech did not adjust himself quite so well as deaf mutes had done in the past with their sign language. The present-day child, having experienced a higher degree of social contacts, expected better things of the community than he received later in life. Where this development was reached difficulties arose later because of the lack of adequate opportunities for work.

DR. M. L. POWELL (Melbourne) said that perhaps he had missed some of these cases from time to time, and therefore he asked whether there was a fairly simple method of screening that could be used in professional rooms that might help to pick them up. Some time previously a mother had brought her baby to him at six weeks of age because she had been told by a photographer that the child was deaf. Dr. Powell had asked her on what grounds the photographer thought the child was deaf, and she replied that he had said to the child, "Look at the dicky-bird", and the child had not taken any notice. Dr. Powell had rung bells and whistled and gooed and booed and finally had advised the mother to bring the child back when it was a little older. Before six months had passed she had advised him that she was certain the child could hear. Was there a simple method of testing in such cases?

Dr. V. L. Collins said that the mother might still be wrong and the photographer right, because that was a problem often met. The audiometrist was in for a great deal of work in the future because all doubtful cases would be referred to him. For that reason the system would develop. Obviously, that child could still have some degree of deafness; it might hear some sounds, but not speech. The simplest test one could use in the case of a child who could apprehend and hear obvious sounds was a simple command or instruction, without any gestures at all, such as, "Come over to me", spoken softly and with varying intensity. The older child one could ask to repeat something. That was a simple method. The old ticking watch and the tuning fork were useless in such cases. One did not know what the child could hear, but if one asked the child to repeat something and the child repeated it accurately, or it obeyed some simple instruction spoken softly, one could find out something. Such methods were simple. Some children could read lips and had a good deal of intelligence, and yet had a high tone loss.

Dr. Powell asked whether Dr. Collins agreed that the photographer in the case mentioned was a bit premature.

Dr. Collins replied that he did.

The chairman asked Dr. Davey to say something on that aspect.

Dr. P. Davey said that the baby, according to Mrs. Ewing, responded to familiar sounds not given undue amplification such as the tinkle of a spoon on its bottle. In regard to early pure tone audiometry, mental ability was the determining factor. The bright four-year-old might give an accurate response at the first test, a retarded child might not be capable of this before he was seven years old. By contrast, objective audiometry by the psychogalvanic skin resistance method would yield a pure tone audiogram at threshold from the early months of life. By this method a galvanic stimulus was used as a conditioner for sweat gland reaction. A pure tone stimulus then replaced the electric shock, and audiometry proceeded without subjective cooperation.

DR. DOUGLAS GALBRAITH (Melbourne) said that in the case of children, particularly older children, who had a considerable degree of deafness, one of the first problems was to decide when it was advisable to use a hearing aid. Another problem was the period of education in the use of the aid that was required.

Dr. V. L. Collins said that there were differences of opinion on those subjects. A certain amount of action had to be experimental. The audiometrists relied on the audiogram a good deal. They used hearing aids when the loss was between 25% and 80% and between 30 and 40 decibels up to 100 decibels. It depended a lot on the kind of hearing loss. Again, there were differences of opinion as to the degree on which one should rely on hearing aids and what amount of training was required. In Melbourne they recognized that any child who used a hearing aid required training. There were other aspects of this subject which should be gone into, but Dr. Collins preferred not to do so just then. He would like to hear Dr. Davey's opinion.

Dr. P. Davey said that the subject of hearing aids was full of difficulties. One could not expect a child to respond at once to their use. There might be only a limited improvement of a child's hearing, and then only in some ranges. In Manchester and in the clinics of the United States of America that dealt with young children mostly, they agreed that the child should receive auditory training—training of its ear—with the voice amplified or with amplification at the hands of a teacher before the hearing aid was fitted. Then the child should be tried with the hearing aid with the help of the teacher. The parents should be instructed in the approach to the use of the aids. The child should not merely be given an aid and then sent forth into the world untrained in its use. That should be insisted on at all times. The correct application of hearing aids presented a very big problem to the department which was concerned with the physical side of the matter. Its members could well employ their time improving the apparatus and keeping the mechanical side in good order. There was a great field there. They should realize that they were not educators or medical men or even psychiatrists or psychologists. There were the two separate compartments of this big problem and all of them had to face it. They should avoid impinging on one another's fields. There was a tendency, Dr. Davey feared, on the part of the manufacturers of hearing aids to impinge on the field of others.

DR. R. WALL (Launceston) said that reference had been made to the child in the mildly deaf stage, the child who had had his tonsils and adenoids removed and had retracted ear drums and assorted ear troubles and who might or might not be partly deaf. Dr. Wall had had the good fortune to live in an area where a deep X-ray service had been well developed for many years. Dr. Davey had mentioned the use of radon. Dr. Wall had had a lot of benefit from the use of deep X-ray therapy.

DR. R. A. R. GREEN (Sydney) said that mention had been made of the deaf child who was also blind. These handicaps were almost sufficient to put such children in the mentally affected class although they were not mentally affected, but only mentally retarded. Dr. Green supposed that they all agreed that the deaf child who was blind could be regarded as greatly handicapped. Because of their double disability these children might be regarded as being incapable of being educated. It might interest Dr. Powell to know that there was a type of stethoscope which had a metal back and a celluloid side. One side dealt with the deeper tones and the other side with the higher tones.

DR. F. W. CLEMENTS (Sydney) said that the point that struck him was the importance of the early recognition of

the deafness if they were to expect the deaf child to take its proper place in the community. That placed a great responsibility on the pædiatrician and the obstetrician, more particularly the former. Although these children might not be suitable for hearing aids, there were other things that could be done. Among these was the training of the mother to recognize the child's limitations. Dr. Clements insisted again that the early recognition of the deafness was important. If necessary, the child should be taken to the right place for an audiogram. There should be no delay. If there was delay, and then after five or six years the child was suddenly thrown into the community with a hearing aid, with all its difficulties, the child might be retarded in settling down in its proper place in the community. The aim should be to recognize deafness early and so prepare the child for the consequent retardation that it might suffer. Then they should utilize all the devices that were available.

Dr. H. O. Lancaster, in reply, said that the only additional thing he desired to say was about genetic causes. There had been a swing of opinion in regard to these cases. In the old days people had been content to attribute the trouble to congenital syphilis and a few post-natal causes. Congenital syphilis should not be given as the explanation of everything; more attention could be given to genetic causes. Inquiry should be made among the families of the children affected.

Dr. V. L. Collins, in reply, said that his only further comment was on the ear, nose and throat cases linked with Dr. Davey's remarks that minor grades of deafness were often due to a middle ear or other condition. It would be interesting to know how many cases would have been prevented from becoming severe if they had a better check on children which would reveal deafness earlier.

Dr. J. F. Williams, the chairman, thanked the principal speakers and those who had taken part in the discussion. Dr. Powell had said that he wondered how many cases he had missed. Dr. Williams himself had missed a good number, even with something approaching the knowledge that he now thought he had. They had all missed cases in the past. He hoped they would not miss so many in the future. Probably they were only touching a part of the problem when they thought of the children who were referred to hospital clinics. In the community there must be many more whom they did not see. Reference had been made to the association of the Commonwealth Acoustical Laboratory with the medical profession and pædiatricians and ear, nose and throat specialists. Dr. Williams could only hope that in the future there would be the closest liaison between all concerned. There was no doubt that the laboratory was doing more and more work in the fitting of hearing aids, but he thought that the work required the cooperation of pædiatricians and the ear, nose and throat specialists if it was to be what it should be.

Cross Infection in the Hospital.

A round-table discussion took place on cross infection in the hospital, DR. R. SOUTHBY (Melbourne) in the chair.

DR. KATE CAMPBELL (Melbourne) read a paper entitled "Cross Infection in the Neo-Natal Nursery" (see page 329).

DR. ERIC SIMS (Adelaide) read a paper entitled "The Physician's Worries in Relation to the Older Children" (see page 331).

DR. K. B. FRASER (Brisbane) read a paper entitled "Cross Infection in the Hospital: The Pædiatric Surgeon's Problems" (see page 333).

DR. J. W. PERRY (Melbourne) read a paper entitled "The Contribution of the Laboratory to the Problem of Hospital Infection" (see page 339).

DR. CLAIR ISBISTER (Sydney) said that it was obvious that all present were acutely conscious of their grave responsibility to prevent cross infection in hospital. It was a very serious state of affairs that children with lowered resistance could enter hospital with one disease and contract another; it was probably even worse that healthy babies could become infected in a maternity hospital. However, Dr. Isbister did not feel that it was equally obvious that they had clearly considered the factors that determined whether a person acquired an infection or not. She did not think it was possible to attack the whole problem scientifically in a satisfactory way unless they considered the following factors, namely: (*a*) the resistance of the patient, (*b*) the dose of the organism and (*c*) the virulence of the organism, and considered them separately and together. During the previous four years Dr. Isbister had been studying the infections occurring in a newborn nursery of a maternity hospital that had about 2000 deliveries a year. She had been reluctant to introduce more routine procedures aimed at reducing the dose of the organism and preventing contact spread without knowing more of the organisms causing the infection. As Dr. Campbell has said, routine procedures could be a danger if badly used; they could also be expensive and irritating. In May, 1952, they had had a small outbreak of five cases of hæmolytic *Staphylococcus aureus* infection; two of the babies had a breast abscess and the others pustules that were larger than usual and surrounded by small areas of cellulitis. Breast abscesses in babies were rare and it seemed possible that one particular strain of staphylococcus could have caused the infection, so Dr. Durie and Dr. Isbister had asked Dr. Phyllis Rountree to phage-type the staphylococci. They had proved to be the same strain, much to Dr. Isbister's satisfaction, and Dr. Rountree had offered to phage-type all organisms cultured from infections and to assist them to study the epidemiology of the staphylococcus. They were at present writing up the results of this investigation and some of these might be of interest to those present.

Dr. Isbister could not give exact figures, but during that year hæmolytic *Staphylococcus aureus* had been cultured from over 200 babies and over 30 mothers. One particular strain, 47C/52, the cause of the original outbreak, had caused the infection in over 60 of the babies, and Dr. Isbister found that she could diagnose these clinically with a good degree of accuracy as the lesions were mainly skin infections and either abscesses or pustules with marked surrounding reaction. However, two other strains had occasionally misled her with similar lesions though she did think she could say that those more virulent infections more prone to cause skin infections and mastitis in mothers could be recognized. The majority of the infections in the mothers had also been caused by this organism; the common infection was mastitis and breast abscess, and the infection of the mothers followed that of the babies. After the first outbreak they had isolated all the infected babies and nursed them in strict isolation; they had also cleaned out the nurseries, and for three months the troublesome strain disappeared. However, in September it had recurred, the maternity department was busy, the isolation nursery had been taken for a mother, and the babies had been nursed unsatisfactorily in the general nursery with what isolation was possible in a general nursery. Dr. Isbister supported Dr. Campbell in her statement that it was impossible to convince the powers that be in a hospital that an isolation ward should be empty; as soon as it became empty, it was taken for something else, with the result that infections steadily continued to occur in the nurseries. The nursing staff had been examined for carriers and several were found who were carrying the organism, but they were no longer in the nursery and did not seem to be the main cause of the trouble. Anyway the staff were then considered to be possible carriers, and whereas they had worn masks only in the premature ward, they now had to wear them in the general nurseries. The only result of this was that the infection rate had increased in the following two months. Dr. Isbister did not, however, blame the masks, badly used though they might have been; she thought that probably the warm humid weather was to blame, but masks did not reduce infection. This did not mean that she thought masks should be discarded because people used them badly; they should be taught to use them correctly and certainly use them in the premature and labour wards.

Finally, Dr. Isbister had found that the blankets were not being satisfactorily sterilized, and when they had been sterilized properly the strain disappeared. However, another more virulent strain had suddenly appeared in at least four Sydney maternity hospitals and it seemed to cause even more serious infections and was also penicillin-resistant; Dr. Isbister thought it would cause a lot of trouble and she hoped that they would get rid of it very soon.

Dr. Isbister was sure that they should recognize virulent organisms and treat them early; phage-typing might be of great help in this, but they had to be clinically alert. Dr. Isbister asked Dr. Perry whether in the five cases of staphylococcal pneumonia typing was done as it sounded like a virulent strain. Thrush was rare and was probably introduced by mothers. However, Dr. Isbister knew a hospital that had had it for a good ten years and where she was sure spores could be obtained from the nursery dust. She agreed with Dr. Campbell that routine circumcisions were dangerous and should not be done in maternity hospitals. She was sure that most infections caused by staphylococci in the nursery were hand spread by the nursing staff. To reduce handling of babies they had at the Royal North Shore Hospital stopped routine bathing and also routine eye drops which often irritated the baby's eyes. Dr. Isbister wished that she could say that their mothers were washing

their hands before breast feeding, as she thought this was more important than washing nipples, but so far it meant too much extra work.

DR. G. GODFREY (Perth) reported an outbreak of severe staphylococcal cross infection in which many babies died. In 70% of cases infection had been found to be spread by nurses. He stressed the importance of the medical staff giving a lead in dealing with the problem.

DR. M. T. COCKBURN (Adelaide) said that he was interested to know that nursery infection was present and presenting difficulties in maternity hospitals in other States. A recent epidemic of staphylococcal infections in mothers and infants had occurred in the Queen Victoria Maternity Hospital in Adelaide. Phage-typing had been carried out, and carriers discovered among a number of members of the nursing staff.

A group of potential carriers of infection, not so far mentioned by any speaker, was the medical students. These people came to the wards and nurseries of hospitals with scant thought or knowledge of the correct procedures to adopt in the problem of prevention of cross infection.

It behoved all who were associated with students in their clinical teaching to advise each new group of the dangers of cross infection and of the best practical ways of attempting its avoidance.

DR. D. JACKSON (Brisbane), commenting on Dr. Sims's reference to night nurses, said that it was important that there should be an adequate number of night nurses on duty. Only too often the authorities who arranged such things seemed unable or unwilling to recognize that in a sick babies' ward there was no difference between night and day. Dr. Jackson went on to say that he wondered whether cubicles were essential or even desirable. He had worked in hospitals with and without cubicles, and felt sure that, given the same amount of space and the same number of staff, and above all proper understanding by the staff, results equally good if not better could be obtained without the false sense of security engendered by structural barriers. As an aid to understanding and discipline, he recommended that the compilation and progress of the cross-infection record should be shared and discussed by medical staff, nurses and students. Dr. Jackson then asked Dr. Perry for advice regarding the use of bowel smears and cultures. At Dr. Jackson's hospital it was the practice to take smears from all infants on admission and at regular intervals thereafter, but media were expensive, and he wondered whether this practice might be making unnecessary work for the laboratory. He said that the laboratory technician collecting blood samples was a common offender against the rules designed to limit cross infection.

DR. V. L. COLLINS (Melbourne) wished to support Dr. Jackson in his plea for fewer cells or cubicles. Barriers against infection could become barriers to good personal care. He did not agree with Dr. Fraser that cells or cubicles were necessary for the nursing of babies in gastro-enteritis wards or to prevent gastro-enteritis. That was a hand-borne infection, and the more doors and barriers that were placed in the nurse's way, the more difficult was her task made and the greater was the risk of more personal errors.

Dr. Sims stated that "until we can get new hospitals we must compromise between the ideal and the practical". He did not believe that new hospitals could provide the ideal. Treatment in hospital would always be a compromise between the ideal and the practical. So long as they had so many nurses' and attendants' hands touching the patient and so many noses coming near the patient there would be cross infection. The obvious answer in children's wards as well as the maternity hospital was to reduce the number of noses and hands coming in contact with the child, and that could be done only by using the mother to care for the child. It was not possible, in many instances, however, for the mother to care for the child throughout his stay in hospital. The washing of hands between patients, the wearing of gowns, and barrier nursing—all these provided a barrier against frequent handling and playing with the child as well as against infection. It seemed obvious, therefore, that they had to use the mother more also to meet the emotional needs of the small child in hospital.

Knowledge upon the spread of infection in hospital was still inadequate. If two children were separated by a screen, one had a respiratory tract infection, and later the second developed a respiratory tract infection, was it known by what means the second baby was infected? Was it an air-borne infection from the first baby, was it a hand-borne infection by an attendant, or was it a direct infection from an attendant's nose or throat? Dr. Collins ventured to suggest that they did not know the relative importance of air-borne or attendant-borne infection. In the case of certain virus diseases like measles, it was probably often air-borne. With the common upper respiratory infections Dr. Collins considered that infection from a recently infected attendant's nose or throat was the usual mode of spread. Such conceptions ought to govern the planning of children's wards in hospital.

In the previous year they had discarded the routine wearing of masks by staff attending infants in the Royal Children's Hospital, Melbourne. The main reason was the impossibility of obtaining from the staff the proper use of masks. The sister tutor in the Nursing School had despaired of obtaining proper mask routine from the nurses in spite of careful training. Medical staff members were often the worst offenders and set a poor example to the nurses. The mask, regarded as a symbol of safety, became a danger because its use was abused. It was considered that it increased the risk of hand-borne infection because so many nurses and other attendants were nasal carriers. There was little risk of air-borne infection from a nasal carrier, but there was a much greater risk of hand-borne infection from the frequent handling of masks. Therefore it was considered much more important to discontinue the use of masks and to place increased emphasis upon the washing of hands.

DR. P. HIPSLEY (Sydney) said that one obvious measure at their disposal was to admit fewer children to hospital. In the old days of home surgery healing had been quicker. An intelligent mother, he reminded the gathering, made a very good nurse.

DR. FELIX ARDEN (Brisbane) commented that he was very interested to hear Dr. Kate Campbell's views on the dangers of wearing masks under conditions prevailing in teaching hospitals. He agreed whole-heartedly. He thought that one advantage of cubicles was that they prevented cots being placed too close together, as happened constantly in open wards. In the Brisbane Women's Hospital the cross infection of which they were continually afraid was gastro-enteritis. The prevailing organism was a coli at present, salmonella strains being far less common. It was almost impossible to prevent the periodic introduction of this infection, as it had been found that up to 10% of healthy adults, including mothers and nurses, could be symptomless carriers. Some babies also carried the organisms. As a precautionary measure they had resorted to taking weekly rectal swabs in the premature surgery and frail babies' ward with immediate isolation of carriers. In addition any baby with loose stools was at once removed from the nursery, and if the infection was proven all the babies in that nursery were given chloramphenicol as a prophylactic (50 milligrammes every six hours for three days). Fortunately, the organism was susceptible to chloramphenicol, but there had been three deaths this year nevertheless. In comparison to this, staphylococcal infections were of nuisance value only, limited to occasional "sticky" eyes, paronychia or pustules. There had been no serious staphylococcal infections for some time.

SIR HARRY WUNDERLY (Canberra) spoke of the problem of children of tuberculous mothers isolated in preventoria for B.C.G. innoculation. They were, unfortunately, exposed to the risk of incidental infection. With regard to the isolation of children with the primary tuberculous complex he had been assured that an occasional positive culture from the fasting gastric juice did not constitute a menace. One survey, however, did not support this, and he agreed that those children could easily be a menace to their attendants if they developed a respiratory tract infection with a cough. Sir Harry Wunderly went on to say that he was trying to channel tuberculosis back into the field of general medicine, and to induce teaching and base hospitals to build special wards where the staff could have access to tuberculous cases. By that means knowledge of the disease would spread.

Dr. Campbell, in reply to points raised in the discussion, pointed out that sepsis should be actively treated as well as isolated. She agreed that the incidence of infection was lower in newborn babies who were kept at the mother's bedside, and she knew of two cases of fatal septicæmia after circumcision. She considered it was impossible to stop students and nurses handling their masks, and, supporting Dr. Cockburn's remarks on students as carriers of infection, recommended if it was necessary to use a baby for teaching, that the baby should be brought out to the students rather than that the students should be admitted to the nursery. She agreed with Dr. Hipsley's remarks, but pointed out that small present-day houses and such factors as the forty-hour week often made home nursing difficult.

Dr. Fraser, in reply, said that Dr. Collins had remarked that he, Dr. Fraser, had talked on nursing

gastro-enteritis patients in cells or cubicles. What Dr. Fraser had said was: "An important aim is to nurse all surgical cases in children under one year of age in cells or cubicle wards restricted to surgical cases only." He stood firm in this opinion. The second point was that while it was hard to get nurses and even doctors to wear wasks properly, in certain specialized wards nurses could be taught to wear masks intelligently. The almost complete absence of any cross infection in two private hospitals in the nursing of hare-lip and cleft palate patients over the previous eight years had undoubtedly proved the value of that practice. Finally, whatever the merits and demerits of cubicle wards were, the existence of the partition had a psychological value in that it encouraged the nurse to think of each patient as a unit, and that in itself was a milestone on the road to effective barrier nursing.

Dr. Perry, in reply to Dr. Isbister, said that they had not done phage-typing at the Royal Children's Hospital. To Dr. Jackson he replied that nowhere in Australia were the bacteriological services in connexion with diarrhœal disease as advanced as they were in Brisbane. At the Royal Children's Hospital they had temporarily given up searching for *Escherichia coli* (α coli). His advice would be to use routine swabs if trouble developed.

DR. ROBERT SOUTHBY, the chairman, in summing up the discussion, said that he was sure members would agree that they had received a stimulating and instructive presentation of a most worrying and practical problem by a group of people actively engaged in the work of the hospitals where cross infection caused so many headaches to all concerned.

Dr. Southby considered that out of the exchange of ideas two principal facts emerged: Firstly, it was quite evident that the predominating organism at the present time was the *Staphylococcus aureus* and furthermore that this organism was gradually becoming more resistant to each of the newer antibiotics. With regard to the gastro-intestinal infections it was clear that the salmonella group of organisms caused most of the trouble, and in this respect there was no form of therapy which was specific for this group. Secondly, it was quite apparent from all the speakers that the ultimate responsibility for the management, control and dissemination of knowledge of cross infection in the hospital rebounded in boomerang fashion upon the members of the medical profession themselves.

It was only by a strict adherence to all the principles which they held and by meticulously carrying out the details of them in their work in the hospitals that they could hope to impress on junior medical officers, nurses and all working in these institutions how important was the human factor in introducing and spreading infections in hospitals.

Dr. Southby reminded members of the old saying that cleanliness came next to godliness, and he was sure from what they had heard that in the neo-natal field ultra-scientific cleanliness could result in actually sending the babies "heavenwards" if they overlooked the extreme importance of awareness of the problem on the part of every individual. Perhaps if they were less scientifically "clean" they could keep the babies on earth a little longer.

A B O Blood Groups and Fœtal Erythroblastosis.

DR. ELIZABETH TURNER (Melbourne) read a paper entitled "A B O Blood Groups and Fœtal Erythroblastosis" (see page 340).

DR. S. E. L. STENING (Sydney) said that he had seen eight babies who, he thought, were suffering from A B O incompatibility. In each case the anæmia had been mild and the jaundice moderate; microspherocytosis had been a feature and there had been some increase in osmotic fragility, and reticulocytosis and erythroblastosis had been present. No patient had required treatment. These diagnoses had been made on clinical grounds.

Dr. Stening asked Dr. Turner whether there was any quick clinical test, now available or envisaged, on the lines of the Coombs test, by which a quick confirmation might be made. He also asked if exchange transfusion was required, what type of blood should be used.

Dr. Elizabeth Turner said that there were no tests comparable to the Coombs test. The diagnosis had to be made by early jaundice, and it helped to know whether antibodies had been detected in the ante-natal period. Antibodies should be looked for. Also there was generally some varying anæmia present which helped in the diagnosis. In exchange transfusion they used O4 blood, free of anti-A and anti-B antibodies. Most blood banks were aware of their O4 donors who were free of antibodies to groups A and B. They did not use A and B substances to absorb antibodies as Dr. Jakobowicz was of the opinion that this substance might act as an antigen.

DR. CLAIR ISBISTER (Sydney) asked what were the indications for exchange transfusion.

Dr. Elizabeth Turner said that the first indication was the early development of jaundice—within the first eight to twelve hours. The second important sign was the presence of anæmia as manifested by a lowered cord hæmoglobin content. There were other indications, but the chief was the appearance of jaundice. Anæmia was not the important factor. Hyperbilirubinæmia was of more significance than anæmia. They might effect an exchange when there was a high hæmoglobin level in the presence of rapidly developing jaundice because of the liability to kernicterus in hyperbilirubinæmia.

Dr. Clair Isbister said that she understood Dr. Turner to say that anæmia was not generally marked but was marked in severe cases.

Dr. Elizabeth Turner said that the indications were identical with those in infants dying of Rh disease. Dr. Edith Potter in her book "Pathology of the Newborn" stated that the conditions were the same.

DR. FELIX ARDEN (Brisbane) asked whether the histological appearance of the liver was the same in jaundiced babies dying of A B O incompatibility as in similar babies dying of Rh incompatibility. In both cases the clinical evidence suggested liver damage, probably caused by direct action of maternal antibodies. However, in the table exhibited, A or B factor was acknowledged to be present in liver cells, whereas Rh factor had not been proved to be present.

Dr. Elizabeth Turner said that she had no personal knowledge of the histological changes of the liver, but, according to American literature, they appeared to be the same. Wiener stressed the view that kernicterus in A B O disease was more likely to occur than in Rh sensitization, because it fell more on tissues than on the blood.

DR. KATE CAMPBELL (Melbourne) said that Dr. Turner's work was very significant. There was one test which could be helpful after birth, and that was to demonstrate the immune anti-A and anti-B in the mother's serum. She had seen a premature baby recently who showed early onset of jaundice, and it was suffering from erythroblastosis due to A B O sensitization. It was possible to demonstrate definitely immune anti-A antibodies in the mother's serum; the baby responded well.

Dr. S. E. L. Stening asked how long it took to perform the estimation of the anti-A and anti-B in the serum of the mother.

Dr. Elizabeth Turner said that Dr. Jakobowicz was able to supply them with the result in about four hours, which gave them time if exchange transfusion was necessary.

Tension Emphysema: A Surgical Emergency in Infants.

DR. T. Y. NELSON (Sydney) read a paper entitled "Tension Emphysema: A Surgical Emergency in Infants" written by himself and Dr. Douglas Reye (see page 342).

DR. H. WILLIAMS (Melbourne) said that the notes that he had made showed that in three cases the pathological change was in the bronchus. The American literature on this subject was vague. Dr. Williams did not think one could explain these cases on the basis of bronchial obstruction. That opinion could be open to misinterpretation. Mention had been made of a lesion in some of the bronchi. A number of those cases were explained on that basis. There was some difficulty there. Some time earlier Dr. Powell had asked Dr. Williams to see a patient—a boy of ten years who was now twelve years old. He was an example of what was believed to be a similar case. He had a cough and his general condition had been poor since infancy; he was limited in activity. He had a focal bronchial obstruction of the right lung. A lobectomy was to be performed soon. Dr. Williams was interested in Dr. Nelson's tension cyst in the lung. He had had a similar case. Dr. Williams showed a slide which would emphasize the importance of early diagnosis and early treatment. It related to a baby ten days old. After admission to hospital the baby was greatly distressed; the heart was grossly misplaced. It was decided to put a needle in to compress the lung. It was apparently successful in preventing the depressed condition. The child remained fairly satisfactory during the night, but collapsed in the morning. It was a case of an emergency lower lobectomy. The child recovered and was now well, but he had a poor left upper lobe. His condition was one of acute

dyspnœa and cyanosis. The only way to make certain was by a radiological examination.

Dr. R. Howard (Melbourne), in thanking Dr. Nelson for his paper, said that in the literature, of which there was not a great deal, there was not such an interesting and valuable series of cases, especially cases accompanied by acute infection. He did not think that the literature showed many cases in which the cause for this condition had been found. The absence of cartilage was important. Dr. Howard had discussed this matter with a surgeon in Chicago. He had reported a series of about eight cases. The absence of cartilage had not been noted, probably because none of the patients had died. The absence of cartilage was helpful. So far the matter had been obscure. Dr. Howard had had experience of one case in a child six weeks old—an emergency case. The child was comparatively comfortable at rest but became distressed on crying. A left upper lobectomy was performed and the pathologist examined the specimen, but could find no evidence of cartilage deficiency in the bronchi. Dr. Howard was grateful to Dr. Nelson for his information on that aspect. The discussion showed a considerable advance in pathology.

Dr. Nelson, in reply, said that about one-half of the recorded cases had not produced any definite evidence of a lesion. That was probably understandable, because if one did a lobectomy one might not remove the affected segment. It might be left behind in the upper lobe. Potts and some colleagues had reported four years previously some cases in which they thought a ductus was the cause of the obstruction because it crossed the bronchus. In one case there was some abnormality in the bronchial cartilage. There were about one-half of the reported cases in which no cause of obstruction was found.

Congenital Pitressin-Resistant Diabetes Insipidus.

Dr. W. B. Macdonald (Melbourne) presented a paper on congenital pitressin-resistant *diabetes insipidus*. He stated that the condition was more commonly known as the "water baby" syndrome. Reports of infants suffering from the disorder were remarkably few and all had originated in America. The first report was from Johns Hopkins Hospital by Waring and co-workers in 1945, and subsequently Williams and Henry (Boston) and Dancis and co-workers (New York) had reported cases. A few months previously Yun Chen Kao and Steiner (Chicago) had published a case report.

It appeared that although the term "water baby" was a popular catch term, few Australian pædiatricians had seen one of these infants, or if they had, they might have failed to recognize the syndrome. Dr. Macdonald said that this should not be interpreted as criticism, for the physical features of infants with this disorder were so misleading that even the trained pædiatrician might fail to associate the symptom pattern with uncontrolled water loss.

During the previous five years in Melbourne, three male infants had been diagnosed as suffering from congenital pitressin-resistant *diabetes insipidus*. One infant had been investigated in detail, and it was established beyond doubt that he suffered from the same disorder as that described by American workers. The other two infants were investigated less completely but showed similar clinical and biochemical features. Although the syndrome was not common, it appeared that many "water babies" must have died in Australia unrecognized as such.

Dr. Macdonald said that he intended to describe briefly the physiology of the disorder, to present the clinical features, and to suggest a possible pathological background.

The basic physiological abnormality was renal water loss, the result of inadequate renal tubular reabsorption of water. This abnormality was not due to a deficiency of an antidiuretic hormone of the posterior lobe of the pituitary, but to an inability of renal tubules to react to an adequate titre of hormone. Dr. Macdonald then demonstrated a chart showing the difference in responses of two infants, a "water baby" and a control infant with normal renal function, to the ingestion of water, water with the simultaneous intramuscular injection of pitressin, and various isotonic salt solutions. The results were unequivocal, and showed not only an inability of the patient to react to pitressin even in the absence of osmotic diuresis, but also an inability to retain water when given in an isotonic solution.

The result of this physiological disorder was dehydration which was the basis of many features of the syndrome.

Clinical features enumerated by Waring *et alii* were: (i) onset soon after birth; (ii) an erratic and unexplained fever; (iii) vomiting during the first three months of life; (iv) polydypsia and polyuria; (v) an inability to respond to pitressin; (vi) rapid dehydration if fluids were reduced or withheld; (vii) inability to excrete urine of high specific gravity; (viii) high values for serum sodium and chloride; (ix) persistent constipation; (x) familial incidence; (xi) (?) occurrence in boys only. To these could be added extreme muscle hypotonia, and failure to gain weight.

Most "water babies" presented as weak, hypotonic, nonthriving infants, with occasional episodes of dehydration, usually attributed to vomiting which had been present from the age of a few weeks. Overshadowing all and completely stealing the limelight was a protracted pyrexia of obscure origin. Dr. Macdonald demonstrated a temperature chart from the records of the infant who had been observed and investigated in detail. He said that the physician, faced with this clinical picture, could not be blamed for doing what he invariably did, namely, search to the limit for an infective basis of the pyrexia—by examination of urine, by blood cultures, by agglutination tests, by the taking of skiagrams of every system *et cetera*. All results were either negative or unconvincing. The true cause of the pyrexia was dehydration, and the same temperature chart showed the dramatic fall in body temperature following an increased water intake. Usually this fact was not realized until several weeks or even months of fruitless investigation had elapsed.

The clinical feature which was most likely to reveal the nature of the illness was the fluctuating dehydration depending on water intake. Signs of dehydration could develop between feedings and vanish after a feed, so that medical observers often recorded contradictory findings in successive hours, adding to confusion. Thirst in small infants could be easily overlooked and interpreted as irritability, whilst polyuria was a difficult sign to elicit in a nursing infant.

If suspected, the syndrome could be confirmed by demonstrating a hyperelectrolytæmia of a high order. In all three infants, serum sodium and chloride levels had been more than 20 milliosmols above normal. In one baby the serum sodium content was 180 milliosmols per litre. A pitressin test could be done, approximately 300 millilitres of water being given orally after two units of pitressin intramuscularly. The first chart demonstrated the lack of antidiuretic response shown by the "water babies". A water deprivation test was safe if the infant was carefully observed. Urine concentration was poor. If facilities were available, renal function tests would demonstrate little if any impairment in most cases.

Dr. Macdonald then briefly commented on the pathological background of the condition. Investigations had demonstrated that the essential nature of the disorder was an inability of the distal renal tubules to utilize pitressin as an activator of water reabsorption, although the intrinsic ability of the distal tubular cells to reabsorb water was still present. A disorder of this nature could occur when a high osmolarity of the glomerular filtrate presented a load which the proximal tubules could not handle, resulting in osmotic diuresis. The diffuse renal damage of chronic nephritis could also produce polyuria and pitressin resistance. However, neither of these states existed in "water babies" in the early stages of the illness.

The only other disorder of infancy which provided a similar picture was Lignac Fanconi syndrome or cystine storage disease, in which polyuria and pitressin resistance were prominent features. At the Royal Children's Hospital over the previous two years, two infants proved to have this disease had been studied, in detail, and had exhibited clinical features closely resembling those of the "water babies". All three water babies had died, and their modes of death resembled those of infants with cystine storage disease described by other investigators, notably Fanconi himself and Bickel.

Although resemblances in the features of the two disorders were not realized whilst these infants were alive, post-mortem examinations of two "water babies" had provided strong presumptive but not conclusive evidence that these infants suffered from a disorder which was closely allied to, if not a variant of, cystine storage disease.

Dr. Macdonald concluded by stating that he would be surprised if future investigations did not confirm this.

Dr. S. E. J. Robertson (Sydney) asked whether Dr. Macdonald had any idea why a dehydrated baby should get fever.

Dr. W. B. Macdonald replied that the fever was probably due to a diminution in sweating. An Italian worker Montuori, in 1911, had shown that there was a decrease in sweat gland activity in the presence of hyperosmolarity of the plasma. He demonstrated that the effect on the sweat

glands was neurogenic. It appeared that these children preferred to have a rise in temperature and conserve their water.

DR. R. SOUTHBY (Melbourne) said that these children formed another group that did not thrive. The paper brought them back to the old story of going carefully. They could ask the mother for the child's history, and whether it was very thirsty and constantly wetting. Sometimes it was difficult to obtain sufficient urine to check its specific gravity. If one was patient enough to wait to get enough, and could show that it had a low specific gravity, one had something to go on, and it gave one confidence to send the baby along for investigation.

DR. S. E. L. STENING (Sydney) asked whether Dr. Macdonald could say whether these children secreted a normal or average amount of saliva.

Dr. Macdonald replied that when the babies were dehydrated the mouth was dry; when they were fully hydrated the secretion of saliva was normal.

DR. KATE CAMPBELL (Melbourne) asked when the baby was having milk, was the amount of fluid the same as when he was given water.

Dr. W. B. Macdonald said that the quantities were the same. The volume was the same whether the fluid was milk, water or glucose and water. The volume was considerable.

Dr. Kate Campbell asked whether that would not be the only difference.

Dr. W. B. Macdonald said that he had tried to avoid this aspect because it could open up a wide discussion. He thought that it was largely due to nirtogenous substances in the milk. He thought that something in addition to the loss of fluid aggravated dehydration.

Posterior Fossa Cysts.

DR. M. SOFER SCHREIBER (Sydney) read a short paper entitled "Posterior Fossa Cysts due to Congenital Atresia of the Foramina of Luschka and Magendie". He said that this paper formed a portion of a more detailed paper written in collaboration with Dr. R. D. K. Reye, of the Royal Alexandra Hospital for Children. The paper is to be published in full at a later date, and accordingly only an outline is given here.

Dr. Schreiber said that very few instances of this interesting condition had been recorded in the medical literature, and it was generally spoken of as a rare disorder. There appeared to be only twenty reported cases, yet five cases had been seen at the Royal Alexandra Hospital for Children in the previous five years. He therefore felt that this condition could not be of such great rarity. In the developmental abnormality no opening formed in the roof of the fourth ventricle at its inferior end. The lateral recesses might also remain closed, and a very unusual variety of non-communicating hydrocephalus resulted in which there was an enormous cystic expansion of the fourth ventricle. The inferior and posterior walls and most of the superior wall of the cyst were membranous. The incompletely developed vermis was situated in the anterior portion of the superior wall of the cyst, and the floor of the fourth ventricle was the anterior wall of the cyst. The cerebellar hemispheres bulged into the side walls of the cyst. A frequently associated feature was a failure of descent of the transverse sinuses which remained in their early fœtal position on the inferior part of the parietal bones. Because their impression was moulded on the calvarium it was frequently possible to diagnose the condition from a careful examination of plain X-ray pictures of the skull, especially in older children and adults. He said that whilst the visualization of a high position of the transverse sinus on the skiagrams of the skull was pathognomonic of a congenital posterior fossa cyst, the absence of such a sign by no means denied the possibility of such a condition. The position of the transverse sinuses was normal in four out of the thirteen cases in which it was mentioned. Further, the position of the transverse sinuses might not be visible in the skiagrams, especially in the skulls of infants, where the bone was thin, and the grooves cut by the sinuses were not well marked. Dr. Schreiber gave details of five cases in patients seen at the Royal Alexandra Hospital for Children. One was a previously well child of three years, the others were infantile hydrocephalics. Four patients had a high transverse sinus and one had a normally placed transverse sinus. Four patients had congenital atresia of all three foramina. One patient had patent foramina of Luschka. In this case air injected into the lumbar subarachnoid space had readily entered the ventricles. Dr. Schreiber discussed the significance of this finding. He demonstrated the difficulty in determining by ordinary examination whether the foramina of Luschka were patent or atretic. In his opinion the examination of serial microscopic transverse sections was the most reliable method by which this point could be established. He stated that absence of the *corpus callosum* was an associated abnormality in two of his cases. This was of interest because the *corpus callosum* developed between the third and fifth months, whereas the foramina of Luschka and Magendie opened at about the fourth month.

Appendiceal Colic as a Clinical Entity.

DR. ROBERT SOUTHBY (Melbourne) read a paper entitled "Appendiceal Colic as a Clinical Entity" (see page 344).

DR. R. HOWARD (Melbourne) agreed with Dr. Southby's clinical view. The pain was always situated in the region of the umbilicus. It was always colic or appendicitis. If the pain was not localized, Dr. Howard suspected that something else was the cause. That was the first important point. The next point was that it was not indirectly associated with meals. Sometimes the pain came on before breakfast and sometimes after breakfast. A child might have no physical sign on examination, but it would object to having something tight about its abdomen because the pressure caused pain. The next point was one on which he disagreed with Dr. Southby. He did not think that the radiologist was of any help at all. It was a clinical diagnosis. A radiological examination was not helpful. If the diagnosis had been right, and the appendix was removed, the child had no further pain. Dr. Howard removed all kinds of appendices—those with kinks and otherwise. At times there appeared to be nothing wrong with them; nevertheless the children had no pain after the operation. There had been one or two instances of children who had had post-operative pain. The pain might occur two, three or four weeks later. Those children had now settled down and had no further attacks after the first one or two. Dr. Howard could not explain that, but it was true, and he regarded it as of great importance. Dr. Southby had hit the nail on the head when he said he had to persuade his colleagues to take out an appendix. Many people refused to have their appendix removed, but, in Dr. Howard's opinion, if the pain was troublesome the appendix should be removed. However, he did not believe in indiscriminate appendicectomy.

DR. K. B. FRASER (Brisbane) said that they were all worried about these things because it was extremely difficult in some cases to decide whether the pain was due to appendicitis or not. It was most important to exclude every other possible cause of the pain. When he talked to students Dr. Fraser always made a point of dividing inflammation of the appendix into two types—acute appendicitis and chronic appendicitis. If it was an acute case one was justified in operating, but in chronic cases one was not justified in operating until every possible non-surgical method had been excluded. Very often adjustments in the dietetic regime and the consumption of a reduced quantity of fat might remove the pain. He did not agree with Dr. Southby about the X-ray examination. He did not find it of any value at all. Did Dr. Southby attribute any significance to the report of the radiologist that the patient felt pain when pressure was applied to the appendix? The cases referred to by Dr. Southby were regarded by Dr. Fraser as cases of colic or of inflammation. He did not think that pain on pressure could be a significant feature in a true case of colic. If one pressed hard enough in certain regions one could cause pain. Dr. Fraser did not necessarily disagree with Dr. Southby, but he believed that many of these cases were colic in the appendix. They had to be careful about the post-operative history, which was entirely subjective. Dr. Fraser was convinced that, in some cases, a percentage of the pain was in the mind of the mother and not in the body of the child. He was sceptical about accepting a story of the complete absence of what had been apparently persistent symptoms before the operation. Once an appendix was removed some mothers would take no notice of a child who complained of pain in the region of the appendix. The mother would say: "You are all right; run away. You have no appendix."

DR. E. S. STUCKEY (Sydney) said that it was very interesting to hear the different opinions on the subject. In his opinion the condition was a real clinical entity and the pain was of sufficient intensity to produce pallor and to make the child stop playing and want to lie down. If one was careful in one's selection of cases for operation the after-results were usually very pleasing. Dr. Stuckey could not agree that "the pain was in the mind of the mother" as suggested by Dr. Fraser. He did not agree that there was no tenderness in these cases. He had found consistently

present an area of guarding and mild tenderness in the mid-section of the right rectus muscle which he presumed was a referred tenderness. There might be no tenderness in the right iliac fossa. With regard to X-ray examinations after an opaque meal, Dr. Stuckey believed they were of use in that they helped to exclude certain other diseases, and he had found that on the occasions when the radiologist was able to report evidence suggestive of pathological change in the appendix, such as incomplete filling, the operative findings usually verified the X-ray reports and the patient was subsequently free from symptoms.

DR. FREDA PLARRE (Melbourne) did not think radiologists could give any opinion about an abnormal appendix because that was a region difficult to survey. It was difficult to get at the appendiceal region except by a fairly long process of waiting. Films which were taken at set periods might show the appendix filled in normal examinations. Tenderness over the appendix region was easier to elicit outside the dark room. Any child with a pain in the region of the appendix should be regarded with suspicion.

DR. P. L. HIPSLEY (Sydney) took exception to the remark about the pain being in the mind of the mother. He thought that the mother was easily the best observer of her own child. She knew when there was something abnormal. They should take great care before they turned down the remark of a mother in regard to her child's health. In all these cases tenderness would be elicited if a child was examined on several occasions. After a long experience Dr. Hipsley thought that one mistake that was made was to miss appendicitis in children. It was easily the commonest cause of abdominal pain. He thought that the commonest error that was made was failure to make a rectal examination. He had still to see many cases in which appendicitis was diagnosed in under one week. He had seen many cases of abscess from an appendix, but seldom were they diagnosed in under seven days. Of course, all those patients were operated on. It was really a distinction without a difference. If a child had appendiceal colic it would eventually have appendicitis. In all such cases it was right to operate. Any child that showed recurring attacks of abdominal pain would eventually be found to be suffering from appendiceal colic and later would be found to have appendicitis.

DR. D. G. MCKAY (Adelaide) said that he saw a lot of children medically as well as surgically. Many children who had had their appendices removed were not relieved of their symptoms. He felt that there was no doubt that they had had pain before the operation, and were still having pain. They acquired a pallor that was visible to all. There were a number of children whose pain was probably not organic. Dr. McKay did not know the true nature of their pain. In some cases, after the appendix was removed, by no stretch of the imagination could they say that the appendices revealed pathological trouble. Sometimes they could not determine any reason for the pain. He agreed that there were a number of patients whose condition seemed to be improved after an operation. In many cases there was no need to hurry. If they watched the patient for a long time, and ruled out all other possible causes as much as possible, disappointments would be greatly diminished. There were still a number who felt pain after an operation.

DR. T. Y. NELSON (Sydney) said that there was a disease of childhood with recurring pain which they called "Melbourne colic". Did Dr. Southby recognize such a clinical entity? If so, how did he distinguish it from appendicitis?

Dr. R. Southby, in reply, said that he did not know that Melbourne had that distinction. He quite agreed with what Dr. Howard had said about children referring to pain in the navel, and also that the attack often happened about mealtime, and he felt that it was mechanical. He also agreed with Dr. Fraser that one had to go carefully into the child's history. That was why he gave the examples he did. They had been carefully investigated to exclude all other possible conditions. The onus was on the clinician to exclude other causes of pain. There might be numerous causes. He was glad that Dr. Stuckey was on his side and that he agreed that they did obtain some help from radiologists. He also agreed that it was a clinical diagnosis. If one watched the children for some time and they continued to have pain, an operation was desirable. A further reason why Dr. Southby tried to obtain help from the radiologists was that perhaps the children had been drifting around among two or three doctors, and so he found the radiologist's report a help. It was good to have something in black and white. With all due respect to Dr. Fraser, whose opinion he admired, Dr. Southby thought that the pain was in the body of the child as well as in the mind of the mother. Dr. McKay had referred to the fact that some children still had pain after operation. Dr. Southby submitted that such children were not suffering from the colic that he had described.

An Experiment in Goitre Prophylaxis.

DR. F. W. CLEMENTS read a paper in which he described a five-year experiment in goitre prophylaxis. This paper has not been submitted for publication.

Demonstration of Pulmonary Collapse.

DR. R. N. O'REILLY (Melbourne) showed three boards demonstrating the ætiological grouping, clinical features, and results of treatment in 240 cases of lobar pulmonary collapse studied by the Clinical Research Unit of the Royal Children's Hospital, Melbourne, over the previous five years.

The children were divided arbitrarily into two groups: those with acute collapse, in which the condition had been present less than one month at the time of diagnosis, and those with subacute collapse, in which the lesion had been present for more than one month but less than twelve months. Those presenting with symptoms of more than twelve months' duration were excluded from the series, as by that time permanent collapse and bronchiectasis were almost invariably present, and could not be influenced to any extent by treatment. He said that the numbers did not represent an entirely accurate distribution of the various ætiological agents responsible, as all of the children included were treated as in-patients in the hospital; frequently children with acute collapse were treated from the out-patient department, and these cases were not included.

Acute Collapse.

The first board illustrated acute collapse. It showed the pathological mechanisms involved in pulmonary collapse, and the ætiological grouping and clinical features of the 130 cases studied. The principal mechanism in acute pulmonary collapse was bronchiolar or bronchial obstruction. Bronchiolar obstruction was due to multiple blockage of small bronchi and bronchioles in the periphery of a lobe by secretion and/or inflammatory swelling of the mucosa. Bronchial obstruction was due to a plug of mucus or a foreign body obstructing a lobar or segmental bronchus. Multiple bronchiolar obstruction was the more important. This was made evident by the fact that the commonest ætiological cause of collapse was a diffuse bronchiolitis, and a plug of exudate or mucus was not demonstrated obstructing a bronchus in those examined bronchoscopically. Defective breathing and poor cough reflex as seen in very ill or debilitated children were often aggravating factors.

The ætiological factors responsible for collapse in these 130 children were: non-specific respiratory tract infection in 38%, underlying allergic diathesis in 22%, inhalation of foreign body into the bronchial tree in 12%, anæsthesia and operation in 12%, and pertussis and morbilli in 6%.

Non-Specific Respiratory Tract Infection.—The majority of these children had been quite ill and were usually diagnosed as suffering from pneumonia or diffuse bronchiolitis. Usually collapse was not diagnosed clinically, but was revealed by radiological examination. The lesion had been unilobar in 70% of cases. The right side was affected three times more frequently than the left, and the right middle lobe was most commonly involved. In 40% of the cases pathogenic organisms were grown from sputum or cough swab and half of these infections were due to *Staphylococcus aureus* alone or in combination with β hæmolytic streptococci or *Streptococcus pneumoniæ*. Of the children affected 50% were under two years of age. Resolution occurred in the majority, and recurrence of collapse was very uncommon.

Allergy.—Collapse in the child with allergic diathesis had been usually precipitated by a mild respiratory tract infection or an attack of asthma. The lesion was generally revealed by radiological examination during an attack of allergic bronchitis, or when cough persisted for more than a week or ten days after an attack. In 90% of cases there was a family history or personal past history of allergic manifestation and it was uncommon for collapse to develop during the initial attack of allergic bronchitis. One of the characteristic features of this type of collapse was the frequency with which it recurred, and 50% of these children had suffered from collapse on two or more occasions; usually different lobes were involved in each episode. Lobes on the right side were affected twice as frequently as those on the left, and the right middle lobe was most commonly involved. Over 50% of the children were between two and six years of age. Culture of sputum or cough swab yielded pathogenic organisms in 50% of cases, and the β hæmolytic

streptococcus was the commonest organism grown. Resolution occurred rapidly in most cases.

Foreign Body.—Sixty *per centum* of the children had presented within forty-eight hours of inhaling the foreign body and the remainder within the first two weeks. Lower lobes were almost always involved and the right side more often than the left. Of the children 75% were between the ages of one and five years. Resolution quickly followed extraction of the foreign body and treatment of infection when present.

Post-Operative.—Of the children, 50% had a respiratory tract infection at the time of operation. Onset of collapse was usually evidenced by cough, fever and sputum, but some had sudden onset with dyspnœa. Seventy-five *per centum* of the children were over the age of six years. Collapse usually affected one lobe, commonly the lower and especially the left lower lobe. Resolution was the rule.

Pertussis and Morbilli.—In the acute phase of pertussis and morbilli children were rarely admitted to the Royal Children's Hospital and so the number studied was small. In those treated, collapse was precipitated by pyogenic infection and resolution occurred with adequate treatment.

In discussing these children, Dr. O'Reilly said that only cases of lobar collapse were included in the series and the criteria for diagnosis of collapse were entirely radiological. The collapsed lobe appeared as a fairly uniform radio-opacity of lobar distribution which occupied a smaller volume than normal. This was determined either by changes in position and retraction of the interlobar fissures, by shift of heart and mediastinum to the affected side, by elevation of the diaphragm and crowding of rib spaces, by compensatory emphysema in the remaining lung tissue on the affected side or by any combination of these factors. He said that although the classification was purely arbitrary it gave some guide to prognosis. Although there was some variation, acute collapse resolved completely in the majority of children, while with subacute collapse there was always danger of permanent collapse and lung damage.

Treatment in cases of acute pulmonary collapse was confined to bed rest, adequate chemotherapy, and where possible physiotherapy. Although the impression was that reexpansion would occur in the majority regardless of treatment, a certain number of children had drifted into the subacute phase. Consequently it was essential to have clinical and radiological evidence of resolution before treatment was completed. Except when there was suspicion of the presence of a foreign body in the respiratory passages, there was no indication for bronchoscopic examination in children with acute collapse.

Subacute Collapse.

The second board illustrated subacute collapse and showed the reasons why acute collapse persisted and the ætiological grouping of 111 cases studied.

Acute collapse could persist as a result of continued bronchial or bronchiolar obstruction, persistence of infection, bronchial or parenchymatous damage, or defective respiratory movement. One or a combination of these factors could be the cause in any individual case.

It was essential to consider which of these factors was responsible in any individual child as adequate investigation and treatment were only possible when the ætiological factors were known. Bronchial obstruction due to foreign body, tuberculous bronchial lesion or bronchial stricture could be excluded only by bronchoscopic examination. Persistence of infection could be due to the poor general condition of the child, to a pyogenic infection with organisms resistant to antibiotics or insensitive to those which had been used in treatment, or to a lesion which did not respond to antibiotics or was usually slow to resolve as in tuberculous infection. Consequently sputum culture, *in vitro*, sensitivity tests to various antibiotics and Mantoux testing were indicated.

The ætiological factors responsible for collapse in these 111 patients were: respiratory tract infection in 36%, primary tuberculosis with involvement of bronchi by hilar glands in 36%, *fibrocystic disease of the pancreas in 17%, allergy in* 4%, myopathy or other muscle weakness in 4%, and foreign body in the respiratory tract in 3%. Tuberculous collapse, *fibrocystic disease of the pancreas and the myopathy group* were not included in acute collapse, because, by the nature of the underlying pathological change, these cases almost *invariably became subacute in character.*

Respiratory Tract Infection.—The children with respiratory infection usually presented with general ill health and residual cough following bronchitis, pneumonia, pertussis or measles. Collapse was usually unilateral, frequently unilobar, and the dependent lobes were invariably affected. A number of these children were presented by Dr. Howard Williams in his paper "Subacute Collapse with Pyogenic Infection and Bronchiectasis", and the third board illustrated the details of this paper.

Tuberculosis.—Of children with primary tuberculous infection 2% to 3% suffered from infiltration or ulceration of a bronchus by tuberculous hilar glands. Bronchial obstruction and aspiration of tuberculous caseous material gave tuberculous pneumonitis, pulmonary collapse or a combination of the two. Cough and wheeze due to bronchial narrowing, and constitutional symptoms due to tuberculous infection were the outstanding symptoms. Collapse was usually unilobar; any lobe could be involved, but the right upper and right middle lobes were most commonly affected. Progress was slow as both tuberculous glands and tuberculous collapse were slow to resolve and probably were not influenced by specific therapy. In 85% residual collapse and bronchiectasis occurred. This type of bronchiectasis was dry and symptomless and rarely became secondarily infected with pyogenic organisms.

Fibrocystic Disease of the Pancreas.—Nineteen of 120 children with fibrocystic disease developed lobar collapse. In the majority of children other evidence of the disease such as steatorrhœa, failure to thrive and abdominal distension had been present. In some, pulmonary collapse was the presenting sign and remained so for some months before other symptoms and signs of the disease developed. Of the children with collapse, 70% were under the age of six months. In the majority the collapse was unilobar and in all but two cases the collapse was on the right side. Re-expansion was uncommon as infection persisted; generalized lung suppuration developed sooner or later and eventually caused death.

Allergy.—Only 4% of the children had an allergic basis for subacute collapse, which showed how infrequently acute collapse in this condition failed to resolve. Even in the subacute phase complete resolution was the rule when adequate treatment was instituted.

Myopathy.—Respiratory muscle weakness was not common, but when present was often complicated by pulmonary collapse. Because of failure of the normal cough reflex secretions accumulated in the bronchial tree. Any respiratory tract infection in a child with myopathy, or respiratory paresis due to poliomyelitis or diphtheria, was frequently complicated by collapse. If muscle weakness or paresis was permanent, resolution was unlikely, but if transient, resolution could occur when severe lung damage was not present.

Foreign Body.—In the majority of children a foreign body in the respiratory tract was quickly recognized and promptly removed. Some, when inhalation was not witnessed, presented later with cough and/or wheeze, and a few with hæmoptysis. Secondary infection always occurred if the foreign body was not removed early. After removal of the foreign body this group behaved in the same manner as the respiratory tract infection group.

Special Correspondence.

LONDON LETTER.

FROM OUR SPECIAL CORRESPONDENT.

Group Practice.

A WORKING PARTY set up as a sequel to the Danckwaerts award to general practitioners in the National Health Service recommended the development of group practice, which is broadly defined as several general practitioners working together in close association, doing their work, in whole or in part, in a group of consulting rooms which they own themselves and providing a twenty-four hour service. Official encouragement was given to the development of such practices by the Minister of Health, Mr. Iain MacLeod, after consulting with the representatives of the profession.

An annual sum of £100,000 is to be set aside to provide interest-free loans to doctors for the purpose of acquiring, converting or building surgery premises to be used by a group of three to six doctors, not necessarily in partnership,

but sharing secretarial and other help. Over recent years the idea of group practices has steadily grown, and groups of doctors have already taken the initiative in establishing communal surgeries. The present financial aid is to encourage more to do so. The loans will normally be recoverable from the practitioners' remuneration from the National Health Service.

The *National Health Service Act* of 1948 made provision for the establishment of health centres, but the high initial cost and the dislike that doctors had of renting their surgeries from a health authority and, above all, the feeling that health centres would not come in any reasonable time, have all combined to diminish the early enthusiasm and foster the increase in group practice.

The Work of Nurses.

It cannot be denied that the nurse is an essential member of the medical team. Doctors are inclined to take them for granted, to believe that they will always be there to assist them in their work of treating the sick. The problem of their supply and training, working conditions, accommodation *et cetera* receives little attention from medical men. Nevertheless, the supply and quality of the nurse will affect the standard and scope of medical service in the future.

In England, as in other places, there has been a general shortage of nurses. Hospitals have had to close wards owing to lack of nursing staff. The shortage has been relieved to some extent by recruiting campaigns, by the employment of part-time nurses, and by a form of dilution of labour—namely, the creation of a grade of assistant nurse, State-enrolled, less highly trained than the State-registered nurse, but capable of relieving the trained nurse of simple nursing duties. The General Nursing Council set a high educational standard for the general-trained State-registered nurse, but many women, although by nature excellent nurses, cannot achieve this and must remain assistant nurses, without the hope of promotion to higher nursing posts. The employment of ward orderlies to relieve the nurse of domestic tasks is another common practice in hospitals.

All these measures have enabled the Hospital Service to be maintained and in some instances to make more beds available. But there is evidence that the peak in recruitment has passed, and the problem of providing for the nursing care of the sick has been approached from another angle—that of how to make the best use of the trained nurse. The Nuffield Provincial Hospitals Trust published in 1953 the results of a detailed study of how the work of nurses in hospital wards was being carried out. The main conclusions were that too much of the nursing care of patients (estimated at 75%) was left to student nurses; and once a nurse became fully trained, she had to spend more time on administrative duties than on nursing. Reorganization of ward routine was suggested, but the practical difficulties of this, in those hospitals where the experiment was tried, have been stressed by the matrons. Doctors' rounds come in for some criticism, often occupying the nursing staff for long periods and at awkward times. The Nuffield Job Analysis Report has been studied by the General Nursing Council and their comments have been submitted to the Ministry of Health. The conflict between the economic need to staff the hospitals and the proper education of the nurse cannot be easily or quickly resolved.

Nursing staff shortage is particularly acute in the mental hospitals, and what is graver still, there is a decreasing number of student nurses. The first of a series of recruiting drives for mental nurses was started in November last year. Miss Pat. Hornsby Smith, Parliamentary Secretary to the Ministry of Health, announced that the Minister had made a special allocation of one million pounds for mental health services for 1954-1955, and the first new hospital building in this country authorized since 1938 was to be a mental deficiency institution of 1040 beds. Further schemes for the next five years had been approved. All this would be valueless, unless nursing staff could be recruited. The first task was the education of the public to dispel the old idea of shame and fear in relation to mental ill health. The new methods of treatment had made mental nursing a different job from what it was thirty years before. Today mental nursing could provide a career as satisfying as any other type of nursing.

The Minister of Health has recently appointed two mental nursing officers, a man and a woman, to the staff of the Chief Nursing Officer at the Ministry of Health to deal with staffing problems in mental hospitals.

Other aspects of nursing have also received the Ministry's attention. A working party has been appointed to consider the training of the district nurse, a most valuable member of the domiciliary health team. Between 8000 and 9000 nurses are engaged either full time or part time in district nursing in England and Wales. They receive special domiciliary training mainly under the auspices of the *Queen's Institute of District Nursing in London*, after completion of their general training, and they work under health authorities responsible for the Home Nursing Service.

Another working party has been appointed recently to advise on the proper field of work and the recruitment and training of health visitors in the National Health Service and the School Medical Service. Health visitors are qualified nurses with extra training in midwifery, public health and the social services, who in the past have been mainly concerned in advising mothers on their health and the health of their young children. The National Health Service envisaged a wider role for the health visitors covering the health of the whole family in cooperation with the family doctor and the hospital.

A break with tradition in the life of the nurse is the recommendation of the Ministry of Health's Nursing Advisory Committee that "living in" is not necessary for all nurses. Exceptions are the matron, the sister in charge of an "acute" ward, and student nurses in their first year. The Minister has adopted this recommendation, and in a recent circular to all hospital authorities suggests that nurses, with the above exceptions, should be allowed to live in single rooms or flats of their own choice. Hospital authorities are invited to follow the example of the universities by maintaining a register of approved lodgings and to acquire large houses which can be converted into flats, leaving the nurses to make their own arrangement for catering. Building on the lines of the traditional nurses' home is to be avoided. This change from the cloister to the hearth has economic advantages for the hospital authorities, but also permits a greater sense of independence to nurses and a closer contact with the outside world, leading to a better understanding of the problems of patients.

Blood Transfusion.

Every year over half a million bottles of blood are required in England and Wales by the National Blood Transfusion Service, representing more than 1500 blood donations daily, or one for every minute of the day and night throughout the year. To meet the steadily growing demand, at least 600,000 active donors are needed. To achieve this target, as well as to replace those who can no longer give blood, another 125,000 donors must be found in 1954.

An analysis of 5000 blood donations showed that they were used as follows: for accident patients, 522; for gynæcology and obstetric patients, 984; for medical patients, 1611; for surgical patients, 1683.

A booklet, "Your Blood and How it is Used to Save Life", has been recently produced by the Ministry of Health to answer some of the questions which blood donors often ask.

Air Pollution.

The disastrous effects of the severe fog in December, 1952, which lasted five days and caused the deaths of four thousand people, mostly the very young and the old, in the area of Greater London, has stimulated much discussion and a public demand for remedies. Fogs are not new phenomena in London, but the fog of December, 1952, was made particularly unpleasant by abnormal meteorological conditions. It was as though a great lid had come down over London; the warm lower air as it rose was stopped by cold air coming down. Exhaust fumes from vehicles added their quota of noxious gases. The special name "smog" has been given to this unpleasant combination, which had a high concentration of sulphur dioxide. Atmospheric pollution was widely discussed last winter with the object of taking measures to prevent a repetition of the disaster of 1952. The London Medical Committee (representing the general practitioners) took the initiative early in November with the suggestion that gauze face masks should be supplied under National Health Service prescription. The Ministry of Health thought that this was too easy a solution and would give a false sense of security, but agreed to supply face masks to sufferers from chronic bronchitis and cardiac disease on a doctor's prescription.

The danger to health of atmospheric pollution was also debated in Parliament. In the House of Lords, Lord Amulree (himself a doctor) asked why the present conditions were tolerated. He said that great care was taken to provide the people with clean food and water supply, but nothing was

done about contamination of the air. What was being done to increase the use of smokeless fuel, or for the provision of smokeless zones in towns and cities? All the suggested remedies would cost money, but fog was expensive in its effects on the health of the people and on our cities. He was not impressed by the value of the face mask, which was a very feeble form of protection. Replying for the Government, Lord Selkirk mentioned the progress made in industry in modernizing fuel-burning equipment. He said that about half the smoke came from industry and half from domestic fires. Of the 16,000,000 domestic grates in the country, in only one-quarter was coke used, and the only satisfactory smokeless fuel which could be burnt in old-fashioned grates was too costly for the average householder. The National Coal Board was experimenting with a smokeless briquette for use in ordinary fire-places. Smokeless zones had been enforced in Manchester and Coventry, but it was more difficult in an area like London. It had been suggested that Westminster should be declared a smokeless zone, but this would be ineffective unless the neighbouring boroughs of Fulham and Battersea followed suit. Lord Selkirk thought that the meteorological conditions which produced the fog of December, 1952, were comparatively rare, but he agreed that action was needed to eliminate air pollution. The Government had appointed the Beaver Committee in July to study these questions, and an interim report would shortly be issued.

The medical officer of the London County Council, Dr. J. A. Scott, speaking at a meeting of the National Smoke-Abatement Society, remarked that the North Sea floods aroused the sympathy of the whole country, but the London fog cost more lives and an untold amount of invalidity. The ordinary citizen in home or office or factory could do much to lighten Evelyn's seventeenth century description of "the hellish and dismal cloud of sea-coal which maketh the City of London resemble the suburbs of Hell".

At a discussion held by the Royal Meteorological Society, Dr. S. T. Wilkins, in charge of pollution investigations at the Fuel Research Station, said that the deaths during the London fog could not have been due to the emission of some abnormal poison, because the same physiological effects arose simultaneously over an area of 100 square miles. Dr. N. Oswald, of St. Bartholomew's and Brompton Hospitals, said that the actual changes in the lungs of patients with chronic bronchitis were due not to any direct poison but to an increase of mucus in the air passages, which put much lung tissue out of action.

Smoke pollution is a problem in industrial areas in many countries, and an international conference at Antwerp has been studying possible preventive measures. Each city has its own problems and suggested remedies may not be universally applicable. St. Louis, a large and highly industrialized city in the United States, provides a shining example of what can be achieved by a strong public opinion and vigorous enforcement of preventive measures. Although the measures for smoke elimination were somewhat ruthless, and certainly expensive, the city of St. Louis was transformed from a grimy city, where, on many mornings, people drove to work with their headlights on, to a smokeless city with an almost Mediterranean freshness in the atmosphere.

Smoke problems may not yet affect Australian cities very much, but with the increasing development of industrial undertakings, the evils of atmospheric pollution can be avoided by preventive action on the part of responsible authorities at an early stage.

Renewed Drive Against Diphtheria.

Local health authorities throughout England have been urged by the Minister of Health, Mr. Iain MacLeod, to intensify their campaigns for the immunization of children against diphtheria. Congratulating the authorities on the remarkable progress already achieved, the Minister recorded that whereas in 1944 there were over 23,000 cases and 934 deaths, the provisional figures for the past year were 240 cases and 24 deaths. He said that the object of the campaign remained the same—to secure immunization of not less than 75% of babies before their first birthday. That gave an objective of 498,000 children in the current year. Only 28% of the babies reaching the age of one in 1951 were immunized; the figure rose to 31% in 1952, but in the first half of 1953 it improved only to 31·5%. This the Minister regarded as still a disturbingly low figure, and he went on to say that as elimination of the disease was conditional upon the maintenance of an adequate level of immunization, an intensive effort would be required to secure the full benefits of the campaign. If parents left their children unprotected, there might be a return of diphtheria outbreaks, as had been exemplified recently in the Midlands, where among 78 cases there were six deaths—all in children who had not been immunized.

Since 1940 up to June of last year over ten and a half million children have been immunized.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

SHORTAGE OF FOOD IN THE COLONY.[1]

THE unsuccessful return of the *Supply*[2] cast a general damp on our spirits, for by this time provisions were becoming scarcer than in a blockaded town. The little live flock, which with so heavy an expense, and through so many difficulties we had brought on shore prudence forbade us to use; and fish which on our arrival and for a short time after had been tolerably plenty, were become so scarce as to be rarely seen at the tables of the first among us. Had it not been for a stray Kangaroo which fortune now and then threw in our way, we should have been utter strangers to the taste of fresh food.

Thus situated, the scurvy began its usual ravages and extended its baneful influence more or less through all descriptions of persons. Unfortunately the esculent vegetable productions of the country are neither plentiful nor tend very effectually to remove this disease. And the ground we had turned up and planted with garden seeds, either from the nature of the soil, or which is more probable, the lateness of the season, yielded but a scanty and insufficient supply of what we stood so greatly in need of.

Correspondence.

THE COLLEGE OF GENERAL PRACTITIONERS: NEW SOUTH WALES REGIONAL FACULTY.

SIR: The first annual meeting of the New South Wales Regional Faculty of the College of General Practitioners will be held in the Robert H. Todd Assembly Hall, 135 Macquarie Street, Sydney, on September 24, 1954, at 8.30 p.m. All members of the Faculty will shortly receive notice of this meeting and are urged to attend and express their opinions on the future activities of the Faculty.

This Faculty only came into being on October 30, 1953, and thanks to the interest of a few members on the committee, steady progress has been made. Dr. T. E. Y. Holcombe, as chairman of the Committee for Undergraduate Education, has been very active. He has been greatly encouraged by the friendly interest shown by Professor Ford and other members of the Faculty of Medicine of the University of Sydney in the aims of the College. We have been allotted two lectures this year on preventive medicine, and these will be delivered by a member of the College. We have been granted permission to approach medical students to offer them facilities to learn something about general

[1] From "A Narrative of the Expedition to Botany Bay", by Watkin Tench (1789). From the original in the Mitchell Library, Sydney.

[2] Lieutenant Ball, in command of the *Supply*, had in February, 1788, taken Lieutenant King with a small staff and 16 convicts to Norfolk Island to found a settlement there. During this voyage he discovered and named Lord Howe's Island. "The shore was in many places covered by green turtle, eighteen of which were brought here and proved a seasonable supply to the convicts afflicted with the scurvy, many of whom were in a deplorable condition. The smallest turtle brought from Lord Howe's Island did not weigh less than 150 lb." (John White's journal.) As food and in particular meat was in very short supply in the settlement the same vessel was sent back to the island for the express purpose of catching more turtle. The trip was, however, quite unsuccessful, as no turtle were seen during several days spent there.

practice. This work will not form part of the curriculum, but will be voluntary. Members of the College have agreed to take students at week-ends and show them how general practice is conducted. District nursing associations have agreed to allow students to go into homes with their nurses to give them some idea of the difficulties of treating patients in their houses. Arrangements are being made for students to visit the suburban hospitals at week-ends to enable them to get a more intimate glimpse of casualty work.

The meeting arranged by Dr. V. M. Coppleson, and held at the University of Sydney last May, was very successful and evoked much interest and discussion in the training of students for general practice. Many practical points were raised which may be difficult to implement, but which have the whole-hearted support of the College.

Dr. Colin Warburton, as chairman of the Post-Graduate Education Committee, has also been very active. Arrangements have been made for a week-end course to be held at Dormie House, Moss Vale, from November 5 to 7, 1954, and many members have already decided to attend. Dr. A. M. Saxby, Medical Superintendent of the Mater Misericordiæ Hospital, North Sydney, and Dr. C. J. McCaffrey, Medical Superintendent of the Royal Newcastle Hospital, have agreed to allow members to attend their hospitals for post-graduate study and training. Members will be advised of the facilities being made available.

Dr. G. Aitkens, chairman of the Research Committee, has prepared a circular for members giving details of the proposed activities of the committee. This aim of the College will possibly interest only a small number of our members.

The College aims to work in close liaison with all medical organizations to improve the standard and status of general practitioners. It does not aim to compete with these bodies, but to supplement the work already being done by the British Medical Association, the Post-Graduate Committee in Medicine, the Royal Colleges and the Faculty of Medicine. Being composed of general practitioners, it feels that it can voice the opinion of general practitioners on many matters of academic interest. As I have previously stated, the College has no medico-political aims.

A cordial invitation is extended to all general practitioners to attend the annual meeting. The College in Great Britain has been making solid headway and has been received with much friendship and practical assistance by all medical organizations. Surely there are many general practitioners of high standing in New South Wales and Australia who will be prepared to join the College to help make it a body of high standing and repute.

Yours, etc.,

W. A. CONOLLY,
Chairman, New South Wales Regional Faculty, College of General Practitioners.

Main Street,
Cessnock,
New South Wales.
August 9, 1954.

CONTROL OF SYNTHETIC NARCOTIC DRUGS.

SIR: The Social Committee of the Economic and Social Council of the United Nations Organization, noting that the consumption of synthetic narcotic drugs is steadily increasing, in July, 1954, has drawn attention to the necessity for strict control over the possession, manufacture, import and export of, trade in and use of synthetic narcotics, and has invited all governments to consider the possibility of carrying out a systematic campaign among members of the medical profession with a view to alerting them to the danger of addiction inherent in the use of synthetic narcotics and to the necessity on their part of exercising great care in prescribing such drugs.

As the Commonwealth is a signatory to the 1948 Protocol, which was introduced to effect control over the synthetic narcotics, and in view of the request from the Economic and Social Council of the United Nations Organization, it would be appreciated if you would draw the attention of your readers to this matter.

The undermentioned synthetic drugs with corresponding proprietary names are those which are at present controlled under the regime laid down by the Protocol of 1948. Of these, Amidone, Dromoran, Heptalgin and Pethidine are the only synthetic narcotic drugs being imported into Australia at present.

1-methyl-4-phenyl-piperidine-4-carboxylic acid ethyl ester (in the form of the hydrochloride and known as *Pethidine*, Antiduol, *Avlon*, Centralgin, D-140, *Demerol*, Dispadol, Dodonal, Dolantal, *Dolantin*, Dolantol, Dolaren, Dolarin, Dolatol, Dolental, Dolinal, Dolopethin, [illegible]sal, Dolvanol, Eudolat, Felidin, Gratidina, Isonip[illegible]ne, Meperidin, Mephedine, Pantalgine, Pethanal, Pir[illegible]sal, Precedyl, Sauteralgyl and its salts.

4,4-diphenyl-6-dimethylamino-heptanone-3 (known as *Methadone*, *Adanon*, *Amidone*, Amidosan, Butalgin, Deptridol, Diaminon, Dianone, Dolafin, Dolamid, Dolophine, Dorexol, Heptadon, Heptanal, Hoechst 10820, Ketalgin, Mecodin, Mephenon, Miadone, Moheptan, *Physeptone*, Physopeptone, Polamidon, Symoron, Synthanal, Turanone) and its salts.

6-morpholino-4,4-diphenyl-3-heptanone (known as Phenadoxone, CB-11, Hepagin, *Heptalgin*, Heptalin, Heptazone) and its salts.

3-hydroxy-N-methyl morphinan (otherwise identified by the symbol NU-2206 and known as *Dromoran*, *Methorphinan*) and its salts.

4-(3-hydroxyphenyl-1-methyl-4-piperidyl ethyl ketone hydrochloride (known as Cliradon, Keto-Bemidone) and its salts.

3-methoxy-N-methylmorphinan and its salts.

1-methyl-3-ethyl-4-phenyl-4-propionoxy piperidine (otherwise identified by the symbol NU-1932) and its salts.

1-methyl-4-metahydroxyphenyl-piperidine-4-carboxlyic acid ethyl ester (known as Bemidone) and its salts.

6-dimethylamino-4,4-diphenyl-3-acetoxyheptane (otherwise identified by the symbol N.I.H.-2953) and its salts.

6-dimethylamino-4,4-diphenyl-3-heptanol (otherwise identified by the symbol N.I.H.-2933 and known as Methadol) and its salts.

α-1,3-dimethyl-4-phenyl-4-propionoxy piperidene (otherwise identified by the symbol NU-1196 and known as Alphaprodine, Nisentil, Nisintil) and its salts.

β-1,3-dimethyl-4-phenyl-4-propionoxy piperidine (otherwise identified by the symbol NU-1779 and known as Betaprodine) and its salts.

4,4-diphenyl-5-methyl-6-dimethylamino-hexanone-3 (known as Iso-methadone) and its salts.

3-Dimethylamino-1,1-di-(2-Thienyl)-1-Butene.

3-Ethylmethylamino-1,1-di-(2-Thienyl)-1-Butene.

Yours, etc.,

A. J. METCALFE,
Director-General of Health.

Canberra, A.C.T.,
August 10, 1954.

SCHISTOSOME DERMATITIS IN SALT WATER.

SIR: Macfarlane, in this journal (M. J. AUSTRALIA, May 17, 1952, page 662), reported outbreaks of dermatitis in Australia which had the characters of schistosome dermatitis, also known as bathers' itch or swimmers' itch. He was able to show that forktail cercariæ developing in freshwater snails from the Murray swamps would produce typical lesions when placed on the skin. The cercaria was *C. parocellata* and the snail host *Limnæa lessoni*, a species common on vegetation in stagnant pools. The vertebrate host of the adult schistosome is not known, but wild ducks and swans are suspected.

The distribution of bathers' itch as reported by Macfarlane included Narrabeen Lake close to Sydney. The salinity of this lake is usually close to that of sea water, but it drops temporarily after rain. The molluscs do not include fresh water species, but are the same as those found in estuaries and salt lakes of the coast. Forktail cercariæ have been found in only one species, *Pyrazus australis*, a small whelk of the shallow sand flats. The cercaria differs from *C. parocellata* in important morphological characters. When applied to the skins of four persons, two of them developed papules at the site and the train of signs and symptoms typical of schistosome dermatitis. Again the host animal of the mature schistosome is not known, but it is thought to be the silver gull.

Bathers' itch has also been reported from Lake Tuggerah, and it is probably common on lakes of the eastern coast of

New South Wales during the summer months. Macfarlane, in the article quoted above, asked for information which would map the distribution of schistosome dermatitis in Australia. Practitioners who suspect that this type of dermatitis occurs in their district are asked to communicate with Professor Macfarlane (University of Queensland) or with the Director of this School.

Yours, etc.,

A. J. BEARUP.

School of Public Health and Tropical Medicine,
University of Sydney,
August 13, 1954.

WRITING TO THE DAILY PRESS.

SIR: Might I endorse the sentiments expressed by Dr. McKellar in his letter "Writing to the Daily Press" in your issue of August 14. I think he expresses what a majority of the profession, both general practitioners and specialists, are thinking. It is quite evident that the profession will have to stiffen its attitude to the hospital problem and such things as treatment by insurance companies. Not only are the latter, on the whole, petty and parsimonious in their payments, but also very slow to settle. Were schedule "G" to include a penalty for delay on the companies' part, then perhaps the position would be different. The time when the profession can be reticent in stating its case on financial matters belongs to the past, not the present.

Yours, etc.,

J. C. JENNINGS.

142 Church Street,
Mudgee,
New South Wales.
August 16, 1954.

CORONARY OCCLUSION.

SIR: It is a pity that coronary failure has come to be called so often "coronary occlusion", since so much of the former is not in any way associated with the latter. I need mention only the well-known fact that so much hypertensive heart failure is a "relative ischæmia", and most of it is not associated with coronary atheroma or occlusion.

After examining many thousands of hearts, I agree with Dr. G. C. Willcocks on the question of exertion. About 1933 I went through a series of very careful records of at least 300 cases of sudden cardiac death. The large majority died in their sleep or while sitting in a chair or walking about a house. In my series, the number dying while undergoing more than average exertion was about 15%. This figure was confirmed soon after by two workers in *The Journal of Industrial Hygiene*. I have a lot of material to support this view, but, obviously, your correspondence column is not the place for it.

I would like to suggest, however, that we drop the terms "coronary occlusion" and "coronary atheroma", which are purely pathological, and adopt the term "coronary failure", which will cover everything including the failure of hypertensive hearts due solely to coronary ischæmia without atheroma or other obstruction.

Yours, etc.,

J. V. DUHIG.

Wickham Terrace,
Brisbane,
August 16, 1954.

A CASE FOR DIAGNOSIS.

SIR: I would like to report the following case and see if any comments are made.

A carpenter, aged twenty-eight years, while at work was knocked by a fluorescent lamp on the head. He combed out the glass and said he thought there had been a trace of blood. This was on Friday. On Wednesday he complained that in the early morning he woke up feeling hot and irritable, and he noticed that he had a rash. On examination he had a very marked urticarial rash on the body, neck and legs. The feet were swollen. There were large spots like giant hives and also large flat raised areas. There appeared to be slight dyspnœa. Adrenaline, minims five, was given and "Ephedro-barbital" tablets four-hourly. The condition was still present next day. "Antistin" (Ciba) was given with good result, also a calcium, ephedrine and phenobarbitone mixture. He complained of an exhausted feeling, substernal pain and tightness of the chest for some days. He stated that he had done no gardening and had eaten nothing different from usual.

The point of interest is that beryllium (which, I believe, is used in light tubes) is known to cause (i) granuloma and (ii) acute dermatitis. Is this a case of beryllium poisoning? I have not read a record of cases of beryllium poisoning with glass, although several articles warn of the danger of cuts from broken beryllium fluorescent tubes.

Yours, etc.,

PERRY HAM.

"Bentham",
333 Glenferrie Road,
Malvern,
Victoria.
Undated.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held at the New Medical School, Frome Road, Adelaide, on July 2, 1954.

Virology.

A symposium was presented on the subject of "Some Aspects of Virology". The chemical aspects were dealt with by DR. R. J. BEST, the biological aspects by DR. J. A. R. MILES, the clinical aspects by DR. J. COVERNTON, and the genetical aspects by PROFESSOR D. J. CATCHESIDE.

Dr. Best quoted Bawden's definition of a virus as "the name for the cause of a transmissible disease with no visible cause". He said that many viruses had now been isolated in what was considered a "pure" state. In all cases the viruses were found either to be nucleoproteins or to contain nucleoprotein as an essential part. Intrinsically, bacterial, plant and animal viruses were alike.

Dr. Best showed tables setting out the gross elemental composition of some fourteen viruses, the nucleic acid content of representative samples of animal, plant and bacterial viruses, the amino-acid composition of strains of tobacco mosaic and influenza viruses, and the relative nucleotide composition of ribo-nucleic acid separated from a number of plant viruses. He then drew the conclusion that differences in virus type were associated with differences in nucleic acid content, and differences between strains of one type were associated with differences in amino-acid composition; however, the nucleic acid in a virus-like turnip yellow mosaic might consist of twenty to thirty different kinds of chain, thus presenting ample scope for variation. He went on to say that it was also important to know what were the roles of nucleic acid and protein in the two processes common to all viruses, namely, infection and replication (multiplication), the latter process embodying the "genetic memory" of the virus. So far all attempts to separate the nucleic acid and protein had resulted in loss of infectivity. However, there was evidence from work with plant viruses that the nucleic acid was of prime importance in the replication process, and it seemed clear that the irreducible minimum in which the genetic memory resided was either the nucleoprotein molecule or some part of it which included the nucleic acid.

There was still some uncertainty about the mechanism by which the virus multiplied in the cell, but on the evidence available it now seemed clear that once it had gained entry into a cell, the virus used the normal enzymes and other normal cell machinery to engineer the production of more of itself, and that it probably entered the genetic mechanism responsible for protein synthesis in the cell.

Finally, it had recently been discovered that when two or more strains of a bacterial virus entered and multiplied in the same bacterial cell, they exchanged "genes" (or character determinants) in the process; and among the progeny were new strains which differed from the original infecting strains but combined some of the characters of each of them. More recently it had been shown that influenza virus behaved in a similar way, and in Dr. Best's own laboratory evidence had been obtained which convinced them that the same process operated when plants were mixedly infected with strains of a plant virus (tomato spotted wilt). This process, common to bacterial, plant and animal viruses, could form

the basis of variation, survival by adaptation and genetic behaviour of viruses.

Dr. Miles said that viruses, although greatly varied in size and structural and chemical complexity, had certain characteristics in common, owing to their intracellular habitat. All viruses were obligate intracellular parasites, incapable of multiplication apart from living metabolizing host cells, whose enzyme systems they must distort to suit their own needs. Their presence necessarily upset the host cell to a greater or less degree, and their prolonged survival necessitated the transference of the active particle from an infected to a susceptible host.

Viruses might be spread by direct contact, and certain of such viruses (for example, that of *herpes febrilis*) could survive for the lifetime of the host, and infect the majority of the host species from infancy to the grave. Others, like the virus of influenza, which was spread directly from subject to subject and could last only for a short time in any one host, might survive in a partially immune population in a form with a low avidity for antibody. Many viruses had a complex cycle with intermediate arthropod hosts. This was true of both plant and animal viruses, and in both cases multiplication might occur in the insect vector and the plant or warm-blooded host.

Dr. Miles concluded by saying that in any discussion on the nature of viruses, their complex behaviour suggesting prolonged periods of adaptation to their present mode of existence and similarities to more highly organized and larger parasites needed emphasis.

Post-Graduate Work.

MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

PROGRAMME FOR SEPTEMBER.

Week-End Refresher Course at Mildura.

THE following programme has been arranged for a week-end refresher course at Mildura on September 18 and 19: Saturday, September 18: 2 p.m., Dr. E. S. R. Hughes, "Ano-Rectal Surgery"; 4 p.m., Dr. John G. Hayden, "Hypertension in General Practice". Sunday, September 19: 10 a.m., Dr. Margaret Mackie, "Recent Advances in Practical Obstetrics, Including Analgesia"; 11.30 a.m., Dr. John Summons, "Common Dermatological Problems".

Fees for this course are at the rate of 15s. per lecture, but those who have paid an annual subscription to the Committee may attend free of further charge. Lectures will be held at the Mildura Base Hospital. Dr. P. G. Barker, Red Cliffs, is the local secretary for this course.

Demonstration at Flinders Naval Depot.

On Wednesday, September 8, at 2.30 p.m., Dr. R. M. Biggins will speak on "Modern Trends in Therapeutics" at Flinders Naval Depot by arrangement with the Royal Australian Navy.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course at Coff's Harbour.

THE Post-Graduate Committee in Medicine in the University of Sydney, in conjunction with the Eastern District Medical Association, will hold a week-end course at Coff's Harbour on Saturday and Sunday, September 25 and 26, 1954. The programme is as follows: Saturday, September 25: 2.15 p.m., registration; 2.30 p.m., "Tuberculous Infection during Childhood", Professor Lorimer Dods; 4.15 p.m., "Surgical Aspects of Diseases of the Chest in Childhood", Dr. T. Y. Nelson. Sunday, September 26: 9 a.m., annual general meeting of the Eastern District Medical Association; 11.30 a.m., "Paediatric Problems—Medical", Professor Lorimer Dods; 2 p.m., "Paediatric Problems—Surgical", Dr. T. Y. Nelson. The fee for attendance at the course will be £3 3s. Those wishing to attend are requested to notify Dr. Angus McNeil, Honorary Secretary, Eastern District Medical Association, 89 River Street, West Kempsey. Telephone: Kempsey 194.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 44 and 47, of July 22 and August 5, 1954.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve.

Appointment.—Leo Kevin Maginnity is appointed Surgeon Lieutenant, dated 12th May, 1954.

AUSTRALIAN MILITARY FORCES.

Regular Army Special Reserve.

Royal Australian Army Medical Corps.

VX700357 Honorary Captain W. B. Fleming is appointed from the Reserve of Officers, and to be Captain and Temporary Major, 11th June, 1954.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical).—1/61767 Lieutenant-Colonel K. J. J. Dorney, D.S.O., relinquishes command of 9th Field Ambulance, 20th June, 1954, and is transferred to 31st Infantry Battalion (1st Military District), 21st June, 1954. 1/61806 Lieutenant-Colonel M. R. Gold is absorbed in a vacancy within the authorized establishment of Lieutenant-Colonels, and to receive pay and allowances of that rank, and is appointed to command 9th Field Ambulance, 21st June, 1954.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—2/146527 Lieutenant-Colonel J. F. C. C. Cobley relinquishes command of 1st Field Ambulance, 30th June, 1954, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District), 1st July, 1954. 2/146556 Major A. G. Finley is appointed to command 1st Field Ambulance, and to be temporary Lieutenant-Colonel, 1st July, 1954.—(Ex. Min. No. 133—Approved 26th July, 1954.)

2/127884 Captain (provisionally) G. B. Smith relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps) (Medical) (2nd Military District) in the honorary rank of Captain, 7th May, 1954.

The resignation of 2/116133 Captain (provisionally) G. E. Cummins of his commission is accepted, 2nd June, 1954.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—3/50196 Major C. D. Donald is appointed to command 6th Field Ambulance, and to be Temporary Lieutenant-Colonel, 1st July, 1954.

To be Captains (provisionally), 25th June, 1954: F3/1019 Pamela Beatrice Blackall and 3/101831 Cameron William Baird.

3/101815 Major G. W. Cooper is seconded whilst in the United States of America, 9th June, 1954.

Central Command: Fourth Military District.

Royal Australian Army Medical Corps (Medical).—To be Captains (provisionally): 4/32051 Robert George Evans, 21st June, 1954, and 4/32054 Lothar Clemens Hoff, 28th June, 1954. 4/32055 Captain R. Hunter is appointed from the Reserve of Officers, 21st May, 1954. To be Temporary Major, 11th June, 1954: 4/32055 Captain R. Hunter.

Western Command: Fifth Military District.

Royal Australian Army Medical Corps (Medical).—The provisional rank of 5/39280 Captain E. A. Smith is confirmed.

5/39280 Captain E. A. Smith is transferred to the Reserve of Officers (Royal Australian Army Medical Corps) (Medical) (5th Military District), 11th May, 1954.

The provisional appointments of the following officers are terminated, 14th November, 1953: Captains 5/26397 W. I. Gordon, 5/26398 H. S. Cohen and 5/26459 V. T. White. To be Captains (provisionally), 15th November, 1953, and be seconded for post-graduate studies in the United Kingdom:

5/26397 William Ian Gordon, 5/26398 Henry Sweetman Cohen and 5/26459 Victor Thompson White. The provisional ranks of the following officers are confirmed: Captains 5/10736 G. M. Nunn and 5/10586 N. H. M. Colyer. 5/26524 Captain (provisionally) R. F. Forkin relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (5th Military District) in the honorary rank of Captain, 19th May, 1954. 5/26526 Honorary Captain P. A. Limbers is appointed from the Reserve of Officers, and to be Captain (provisionally), 5th April, 1954. To be Captain (provisionally), 18th June, 1954: 5/26527 Denis Graham Kermode.

Tasmania Command: Sixth Military District.

Royal Australian Army Medical Corps (Medical).—6/15338 Lieutenant-Colonel C. W. Clarke relinquishes command of 12th Field Ambulance, 30th June, 1954, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (6th Military District), 1st July, 1954. 6/15385 Major L. N. Gollan is appointed from the Reserve of Officers, is appointed to command 12th Field Ambulance, and to be Temporary Lieutenant-Colonel, 1st July, 1954.—(Ex. Min. No. 130—Approved 26th July, 1954.)

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

1st Military District.—To be Honorary Captains, 10th June, 1954: George Lionel Gray, Graham Alan Cossins and Clive Logan Dart.

2nd Military District.—To be Honorary Captain, 31st May, 1954: Harold Thomas Gage.

3rd Military District.—To be Honorary Captains: John Reed Hankey and Neville John McCarthy, 26th April, 1954, Peter Gus Castran, 1st May, 1954, and William Brian Fleming, 10th June, 1954. The resignation of Honorary Captain K. S. Crowley of his commission is accepted, 14th April, 1954.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force: Medical Branch.

John Joseph Bain (0210563) is appointed to a short-service commission, on probation, for a period of twelve months, 1st February, 1954, with the rank of Flight Lieutenant.

The following officers are granted acting rank as indicated, 3rd May, 1954: (Wing Commander) Squadron Leader H. T. Hardy (036451), (Squadron Leader) Flight Lieutenant R. G. Sharp (023027).

Flight Lieutenant A. I. Lane (0210562) is transferred from the Reserve and appointed to a short-service commission on probation for a period of twelve months, 5th May, 1954, with seniority as from 5th May, 1953.

Bede Augustine Hintz (014532) is appointed to a short-service commission on probation for a period of twelve months, 7th June, 1954, with the rank of Flight Lieutenant.

Air Force Reserve: Medical Branch.

The following former officer is appointed to a commission, 13th May, 1954, with the rank of Flight Lieutenant: J. B. Curtis (257233).

Flight Lieutenant H. R. Hawkins (251777) is placed on the Retired List, 16th March, 1954.

The appointment of Flight Lieutenant J. N. Burgess (257884) is terminated, 1st December, 1953.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following have been registered, pursuant to the *Medical Practitioners Act*, 1938-1950, as duly qualified medical practitioners: Asten-Reed, Raymond William, M.B., B.S., 1954 (Univ. Sydney); Bedford, Roma Margherita, M.B., B.S., 1954 (Univ. Sydney); Brighton, William Harold, M.B., B.S., 1954 (Univ. Sydney); Carruthers, Raymund Gerrard, M.B., B.S., 1954 (Univ. Sydney); Clark, Rodney James, M.B., B.S., 1954 (Univ. Sydney); Dawson, Ian Henry Edward, M.B., B.S., 1954 (Univ. Sydney); Golomb, Minna, M.B., B.S., 1954 (Univ. Sydney); Hanley, Brian Robert, M.B., B.S., 1954 (Univ. Sydney); Houston, Henry Thomas, M.B., B.S., 1954 (Univ. Sydney); Keogh, Herbert John, M.B., B.S., 1954 (Univ. Sydney); Leong, Leonard Joseph Aimone, M.B., B.S.,

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 7, 1954.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	8(4)	5(2)	3(3)	..	3(3)	..	..	..	19
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	3(3)	..	..	..	..	..	3
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	2(1)	..	..	..	..	..	..	..	2
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	2(1)	..	..	..	..	..	..	..	2
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	3	12(12)	8(8)	..	..	..	..	..	23
Diphtheria	5(4)	5(5)	1(1)	..	1	..	..	..	12
Dysentery (Bacillary)	..	..	2(2)	..	..	..	..	..	2
Encephalitis	..	1(1)	..	2(1)	..	..	..	..	3
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	14(9)	16(6)	..	..	4(3)	..	1	..	35
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	9	..	..	..	..	..	9
Malaria	..	1	1(1)	..	1	..	..	..	3
Meningococcal Infection	3(1)	4(2)	1	1	..	..	..	..	9
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	1(1)	..	..	..	..	..	..	..	1
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	3(1)	18(10)	..	..	..	..	..	..	21
Puerperal Fever	..	..	1(1)	..	..	..	..	..	1
Rubella	..	8(6)	..	..	4(4)	..	..	..	12
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	8(6)	16(13)	2(2)	8(6)	1(1)	..	..	..	35
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	18	..	..	..	18
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	32(25)	21(15)	15(4)	4(3)	13(11)	5(3)	..	..	90
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

1954 (Univ. Sydney); McMillan, Bruce, M.B., B.S., 1954 (Univ. Sydney); Macken, Thomas Francis, M.B., B.S., 1954 (Univ. Sydney); MacMahon, Edward Brian, M.B., B.S., 1954 (Univ. Sydney); Muller, John Noel, M.B., B.S., 1954 (Univ. Sydney); Murdoch, Wallace Alexander, M.B., B.S., 1954 (Univ. Sydney); Nolan, Kenneth Ernest, M.B., B.S., 1954 (Univ. Sydney); Nordon, Jeanette, M.B., B.S., 1954 (Univ. Sydney); O'Halloran, John, M.B., B.S., 1954 (Univ. Sydney); Oliver, Richard Dalley, M.B., B.S., 1954 (Univ. Sydney); O'Neill, Gerald Allen, M.B., B.S., 1954 (Univ. Sydney); Peirce, Paul Aston, M.B., B.S., 1954 (Univ. Sydney); Philip, George Blackmore, M.B., B.S., 1954 (Univ. Sydney); Pickard, Beda, M.B., B.S., 1954 (Univ. Sydney); Radford, Nancy Vina, M.B., B.S., 1954 (Univ. Sydney); Ramsay, Charles William, M.B., B.S., 1954 (Univ. Sydney); Rawle, John Glanville, M.B., B.S., 1954 (Univ. Sydney); Searle, Alan William, M.B., B.S., 1954 (Univ. Sydney); Shaw, Patrick Lalor Rodas, M.B., B.S., 1954 (Univ. Sydney); Shortland, Gordon Campbell, M.B., B.S., 1954 (Univ. Sydney); Singh, Harold Sylvester, M.B., B.S., 1954 (Univ. Sydney); Symes, Robert Matthew, M.B., B.S., 1954 (Univ. Sydney); Thorburn, Norman Victor, M.B., B.S., 1954 (Univ. Sydney); Treloar, Brian Tremayne, M.B., B.S., 1954 (Univ. Sydney); Vane, Amory Arthur Joseph de Courcy, M.B., B.S., 1954 (Univ. Sydney); Wajeman, Maurice, M.B., B.S., 1954 (Univ. Sydney); Walker, John Edward, M.B., B.S., 1954 (Univ. Sydney); Wallace, Mary Helen Katherine, M.B., B.S., 1954 (Univ. Sydney); Henderson, Bruce Leslie, M.B., B.S., 1954 (Univ. Sydney); Arthurs, Gaston Napoleon, M.R.C.S. (England), 1939, L.R.C.P. (London), 1939, M.B., B.S., 1939 (Univ. London), F.R.C.S. (England), 1951; Crooke, Norman Keith, M.B., Ch.B., 1945 (Univ. Manchester); Kelly, Leo James, M.B., B.S., 1950 (Univ. Queensland); Langan, George Browne, M.B., B.S., 1951 (Univ. Queensland); Linn, Howard Wadmore, M.B., B.S., 1944, M.D., 1954 (Univ. Adelaide); Rhodes, Francis Alexander, M.B., Ch.B., 1951 (Univ. Cape Town); Sandeman, Thomas Fraser, M.B., Ch.B., 1950 (Univ. Edinburgh); Willan, Philip Bertram, M.R.C.S. (England), 1937, L.R.C.P. (London), 1937; Wilson, Margaret, M.B., Ch.B., 1945 (Univ. Glasgow), D.C.H., R.C.P. and S. (England), 1949; Baltgailis, Margers Bertold—for practice in the Ivanhoe Region as defined in a Proclamation published in the *Government Gazette* of May 7, 1954; Sarko, Nikolai—for practice in the Premer Region as defined in a Proclamation published in the *Government Gazette* of May 7, 1954

The following additional qualifications have been registered: Cohen, Douglas Harry (M.B., B.S., 1942, Univ. Sydney), M.S., 1950 (Univ. Sydney), F.R.A.C.S., 1950; Harrison, Keith Selwyn (M.B., B.S., 1936, Univ. Sydney), M.R.C.P. (London), 1940, M.R.A.C.P., 1942, D.T.M., 1942 (Univ. Sydney), F.R.A.C.P., 1954; Hartnett, Bruce Stevenson (M.B., B.S., 1948, Univ. Sydney), M.R.C.P. (Edinburgh), 1954; Hatfield, Kenneth Daniel (M.B., B.S., 1946, Univ. Sydney), D.M.R.D., R.C.P. and S. (England), 1952, M.C.R.A., 1953; Nelson, Selwyn Graham (M.B., B.S., 1930, Univ. Sydney), M.R.C.P. (London), 1947, M.R.C.P. (Edinburgh), 1947, M.R.A.C.P., 1939; Nelson, Wilhelmina Frances Loveday (M.B., B.S., 1930, Univ. Sydney), M.R.A.C.P., 1939.

Notice.

SECTION OF PREVENTIVE MEDICINE OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

The next meeting of the Section of Preventive Medicine of the Victorian Branch of the British Medical Association will be held in the Medical Society Hall, 426 Albert Street, East Melbourne, at 4.30 p.m. on Thursday, September 9, 1954. Dr. A. R. Wakefield, plastic surgeon of the Royal Children's Hospital and the Royal Melbourne Hospital, will give an address entitled "Some Thoughts on Preventive Accident Surgery". All members of the Branch are invited to be present.

Nominations and Elections.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Cahill, Arthur Paul, M.B., B.S., 1941 (Univ. Sydney), 193 Macquarie Street, Sydney.

De Wilde, Frans Willem, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1953, Grafton Base Hospital, Grafton, New South Wales.

Deaths.

The following deaths have been announced:

Nairn.—William Archibald Jock Nairn, on August 5, 1954, at Adelaide.

Synan.—Dorothy Florence Synan, on August 10, 1954, at Kew, Victoria.

Thwaites.—Johnstone Leslie Thwaites, on August 11, 1954, at Albert Park, Victoria.

Diary for the Month.

Sept. 1.—Victorian Branch, B.M.A.: Clinical Meeting.
Sept. 1.—Western Australian Branch, B.M.A.: Council Meeting.
Sept. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Sept. 10.—Queensland Branch, B.M.A.: Council Meeting.
Sept. 13.—Victorian Branch, B.M.A.: Finance Subcommittee.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Tasmania: Part-time specialist appointments for the north-west coast of Tasmania.

Editorial Notices.

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Members and subscribers are requested to notify the Manager, The Medical Journal of Australia, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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